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VOLUME XIV NUMBER 3

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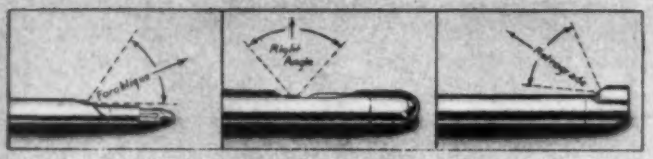

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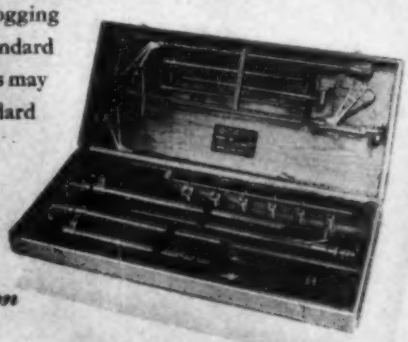

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DISEASES *of the* CHEST

VOLUME XIV MAY-JUNE, 1948 NUMBER 3

The Treatment of Patent Ductus Arteriosus*

WILLIAM S. CONKLIN, M.D., F.C.C.P.**
Portland, Oregon

The surgical treatment of patent ductus arteriosus has been employed more and more widely since Gross reported the first successful case of ligation in 1939. It now appears that we may improve the health and prolong the lives of most children and young adults in whom patency of the ductus arteriosus persists. An increasingly large volume of literature, both in this country and abroad, attests to the fact that this opinion is held by cardiologists and thoracic surgeons alike.

When asked to present a paper on the treatment of patent ductus arteriosus before this meeting of the College, it was my feeling that the subject had been covered more than adequately in the numerous comprehensive articles that have been published. The diagnostic and operative criteria have been well established by the original contributors in this field. It may be of value, however, to review, as briefly as possible, the important contributions that have been made, and to place on record my own small series of cases and the interesting or unusual situations that I have encountered.

Nature and Causes

Anatomical obliteration of the ductus arteriosus frequently does not occur until several weeks or months following birth. According to Christie,[7] 64.7 per cent are still open at two weeks, 44.3 per cent at four weeks, and 12 per cent at eight weeks of age. By the eighth month 98 per cent were found to have closed. Patency existing after the first year of life is more likely to be permanent and to

*Presented at the 13th Annual Meeting of the American College of Chest Physicians, Atlantic City, June 6, 1947.

**From the Division of Thoracic Surgery, University of Oregon Medical School Hospitals and Clinics and the Matson Memorial Hospital for Diseases of the Chest.

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function as a vascular shunt, though obliteration of a patent duct seems, in rare instances, to have occurred in later years.[10,35]

The exact mechanism which results in physiological closure of the ductus arteriosus is still obscure. Kennedy and Clark[24,25] and Kennedy[26] have investigated this problem and a number of hypotheses have been summarized by Mackler and Graham[29] and by Wells.[42] Several factors may be involved. It seems likely that functional closure of the duct generally occurs as soon as the lungs are ventilated.[26] According to Schaeffer's studies[34] the duct gradually becomes obliterated over a period of several weeks as a result of elastic tissue hypertrophy and proliferation which begins during the latter part of fetal life.

In the presence of certain cardiovascular anomalies an associated patent ductus arteriosus serves as an important channel joining the systemic and pulmonary circulatory system. By circumventing an obstructed aorta or pulmonary artery, such a duct may permit blood to reach the peripheral vascular bed of either system. Congenital cardiovascular defects of this character might include the following:

1. Aortic obstruction.
   a. Aortic atresia or subaortic stenosis.
   b. Bicuspid or stenotic aortic valve.
   c. Coarctation of the arch of the aorta.
2. Pulmonary artery obstruction.
   a. Pulmonary atresia.
   b. Pulmonary stenosis (Tetralogy of Fallot).
3. Complete transposition of the great trunks.

Under these conditions patency of the ductus arteriosus supports or permits the continuation of life and an attempt surgically to eliminate the flow of blood through the duct is definitely contraindicated. In some of these cases it may actually be desirable to establish an additional communication between the two systems by producing an artificial ductus arteriosus according to the methods of Blalock and Taussig.[3]

Autopsy series indicate that in over one-third of cases in which a patent ductus arteriosus persists there are no associated cardiovascular anomalies.[1] As obscure as the mechanism of normal physiological closure is the failure of such closure to occur. Gross[16] suggests three possible etiologic factors:

1. The anatomic position and direction of the vessel.
2. A defect in the wall of the vessel.
3. A neuromuscular mechanism (vagus deficiency).

Whatever the conditions responsible for persistent patency may be, it has been estimated by Keys and Shapiro,[27] that there are approximately 20,000 such cases in the United States.

Physiology and Pathology

During fetal life the direction of blood flow through the ductus is from pulmonary artery to aorta (Fig. 1). Following ventilation of the lungs and increase in the pulmonary circulation, the pressure relationships are altered and the blood flow through the ductus is reversed (Fig. 2). Burwell, et al,[5,13] have demonstrated that 45 to 77 per cent of all the blood leaving the left ventricle will pass from the aorta through a persistent patent ductus arteriosus into the pulmonary artery. To compensate for the loss of blood to the peripheral circulation, the volume output

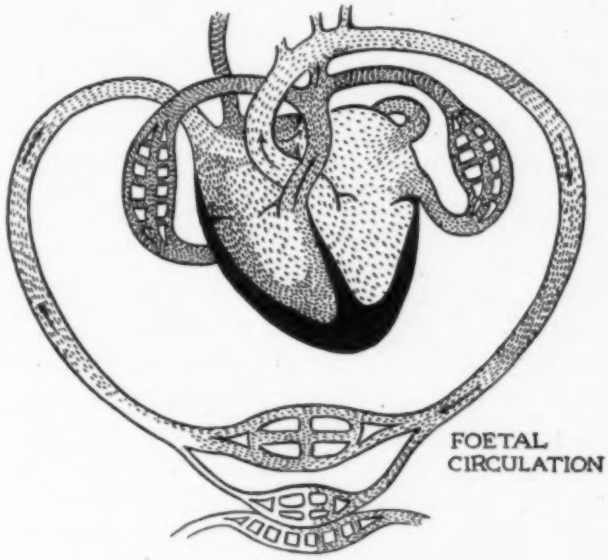


FIGURE 1

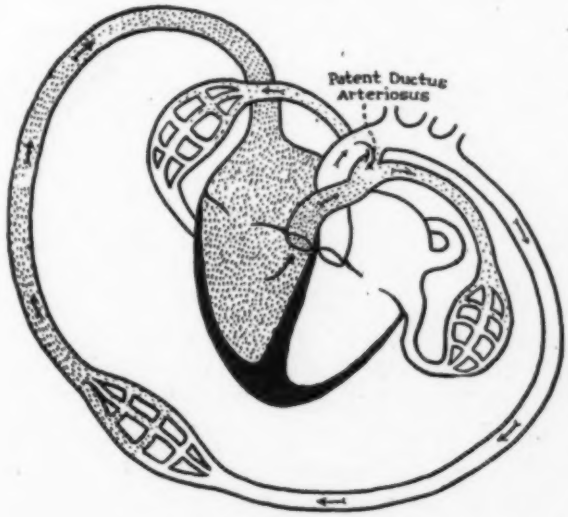


FIGURE 2

of the left ventricle must increase and becomes two to four times as great as the output of the right ventricle. This results in ventricular enlargement and eventually may produce manifestations of left ventricular strain and failure.

The persistently patent ductus arteriosus, then, is an abnormal vascular shunt. While Leeds[28] has pointed out certain differences, the cardiac and hemodynamic manifestations are similar to those associated with an arteriovenous fistula or with aortic regurgitation. The forceful flow of blood from the aorta through the duct into the pulmonary artery sets up traumatising currents and eddies and damages the walls of these vessels producing thickening of the intima, deposition of cholesterol and the formation of atheromatous plaques. Here it is that vegetations of bacterial endarteritis seem likely to occur. Likewise the forceful pressure of blood flowing from the aorta may result in aneurysmal dilatation, or even rupture, of a relatively thin-walled duct.[29] Reduction in peripheral circulation is considered to account for the reduced rate of growth and development of some children in whom the ductus remains patent.

In her classical study of 1,000 cases of congenital heart disease,[1] Maude Abbott found ninety-two cases of simple patent ductus arteriosus. The mean age of death in this group was twenty-four years, however twenty deaths occurred before the age of five. Bullock, Jones and Dolley,[4] eliminating cases that died in infancy, reviewed eighty cases over three years of age and found that 14 per cent had died from their heart lesion at the age of fourteen, 50 per cent at the age of thirty and 71 per cent at the age of forty, only two living into the seventh decade. These and other studies provide ample evidence that a simple patent ductus arteriosus results in a marked reduction in the normal life expectancy.

CAUSES OF DEATH IN CASES OF PATENT DUCTUS ARTERIOSUS

Causes of Death (Cardiovascular)	Abbott (92 cases)	Bullock, Jones and Dolley (80 cases, 3 yrs. old and over)	Shapiro and Keys (60 adults)
Subacute bacterial endarteritis (or endocarditis)	21	42 - 53%	41.7% 6.6% (probable)
Congestive heart failure	24	18 - 23%	28.3%
Sudden death (cardiac?)	16		
Rupture of aneurysm		5 - 6%	6.6%
Heart important factor		4 - 5%	1.7% (suicide)
TOTAL	61 - 66%	69 - 86%	84.9%

The high incidence of premature death, due directly or indirectly to persistence of a patent ductus arteriosus, is illustrated in Chart I, compiled from the publications by Abbott, by Bullock, Jones and Dolley, and by Shapiro and Keys.

It is logical to assume that surgical ligation or division of the patent ductus arteriosus, by eliminating the abnormal shunt, would have prevented or postponed most of the fatalities listed.

Clinical Findings

While twenty of Maude Abbott's cases died before the age of five years, an attempt to diagnose a patent ductus arteriosus in early childhood is rarely made. There is no sure method for determining the presence of a patent ductus in early childhood. It is only in the third and fourth years that the typical clinical findings are likely to become evident.

The symptoms that may be associated with patent ductus arteriosus include the following:

1. Fatigability.
2. Weakness.
3. Irritability.
4. Palpitation on exertion.
5. Pain or discomfort in the left hemithorax.
6. Breathlessness.
7. Syncope.
8. Sensitivity to cold.
9. Epistaxis.
10. Headaches.
11. Hoarseness.

The physical findings include:

1. Subnormal weight and physical development.
2. Pallor.
3. Tachycardia.
4. *Continuous murmur in the pulmonic area.*
5. *Palpable thrill.*
6. *Accentuated or reduplicated pulmonic second sound.*
7. *Increase in pulse pressure with low diastolic pressure* (exercise).
8. "Pistol shot" sound over the femoral arteries (exercise).
9. Corrigan pulse.
10. Capillary pulse.

The ductus arteriosus remains patent about twice as frequently in females as in males. Cyanosis usually does not occur with an uncomplicated patent ductus arteriosus. If it does, it is a manifestation of reversal of flow through the duct. Increased pressure in the pulmonary circulation, as in coughing, crying and respiratory obstruction, may produce transient cyanosis, or cyanosis may appear terminally when the heart begins to fail (*cyanose*

tardive). Clubbing, polycythemia and other evidences of the cyanotic types of heart disease are absent.

Roentgenographically the pulmonary artery appears prominent as seen in the postero-anterior (Fig. 3) and right anterior oblique projections. The heart may be enlarged. Through the fluoroscope one can usually demonstrate increased excursion of the left ventricle, with expansile pulsations of the pulmonary artery and its branches presenting the picture of so-called "hilar-dance."[9] Studies with diodrast ordinarily are neither necessary nor attempted. The ductus itself has not been visualized; but recirculation of diodrast through the ductus, producing prolonged filling of the pulmonary circuit, would be expected and does occur. According to Steinberg, Grishman and Sussman,[37] a localized dilatation of the descending aorta, just beyond the isthmus, can be visualized by angiography. Occasionally angiocardiography is of value in atypical cases in order to rule out associated cardiovascular lesions which would contraindicate surgery.

The electrocardiogram is ordinarily normal. A normal right axis

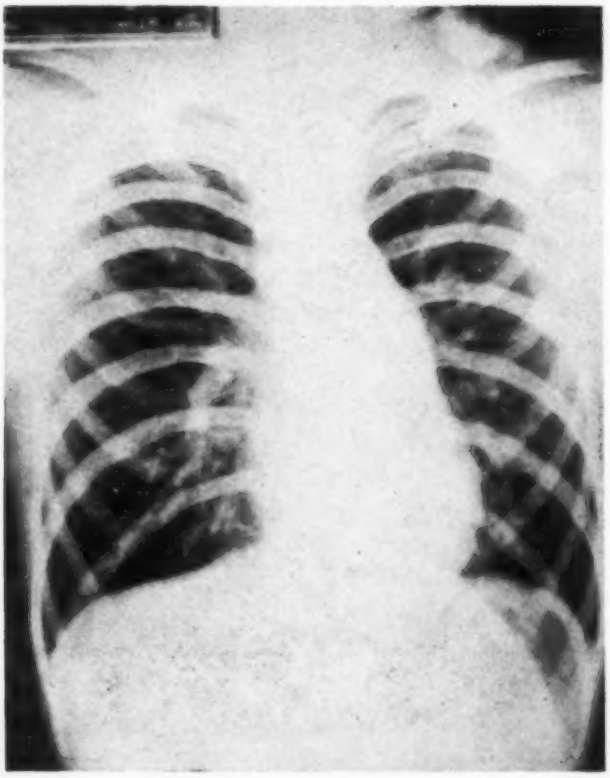


FIGURE 3 (Case No. 8): F.S. Illustrating prominent pulmonary artery segment at upper left border of heart, as seen in PA chest film.

deviation is sometimes seen, especially in the young; but deviation to the left is also consistent with an uncomplicated patent ductus arteriosus. The electrokymograph[21] may prove to be of value, particularly in recording pulsations of the pulmonary artery.

Medical Treatment

An uncomplicated case of persistent patent ductus arteriosus requires no medical treatment. While many of these cases are below average in physical development the majority tolerate normal activity fairly well until complications supervene. Too often anxious parents, apprised of a heart ailment, will overprotect the child. Undue concern and restriction of activities results in psychological damage both to the child and to his siblings.

When congestive heart failure or subacute bacterial endarteritis develop medical treatment must be instituted promptly; but surgical treatment should be given immediate consideration. Bacterial endarteritis complicating a patent ductus arteriosus has been cured by medical therapy.[6,43] Touroff[41] has reported a case which having recovered from subacute streptococcus viridans endarteritis, superimposed on a patent ductus arteriosus, developed a recurrence of the infection twelve and one-half years later and then recovered once more following surgical treatment. Medical treatment certainly provides no safeguard against eventual recurrence nor against the other complications that are prone to occur.

Surgical Treatment

In 1907 Munro[30] reported the isolation of a patent ductus arteriosus at necropsy, demonstrating the feasibility of its surgical ligation. At that time the diagnostic criteria were, it seems, not adequately established and not until 1938 was an ante-mortem attempt to obliterate the duct recorded. Complete obliteration of the duct in this case, reported by Graybiel, Ashton, Strieder and Boyer,[11] was not accomplished and the patient, who had a superimposed bacterial endarteritis, died on the fourth postoperative day with acute dilatation of the stomach.

The first successful case of surgical ligation of a patent ductus arteriosus was reported by Gross and Hubbard[12] in 1939. The brilliant response of this and succeeding cases to surgical treatment has encouraged ever wider application. It soon became evident that the surgical approach to this condition, either by ligation or division, may offer those who have a persistent patent ductus arteriosus the chance for normal survival.

Specific indications for surgery, according to Gross,[16] include the following: 1) Stunting of physical growth. 2) Cardiac embarrassment, 3) Voluntary limitation of physical activity. 4) Evi-

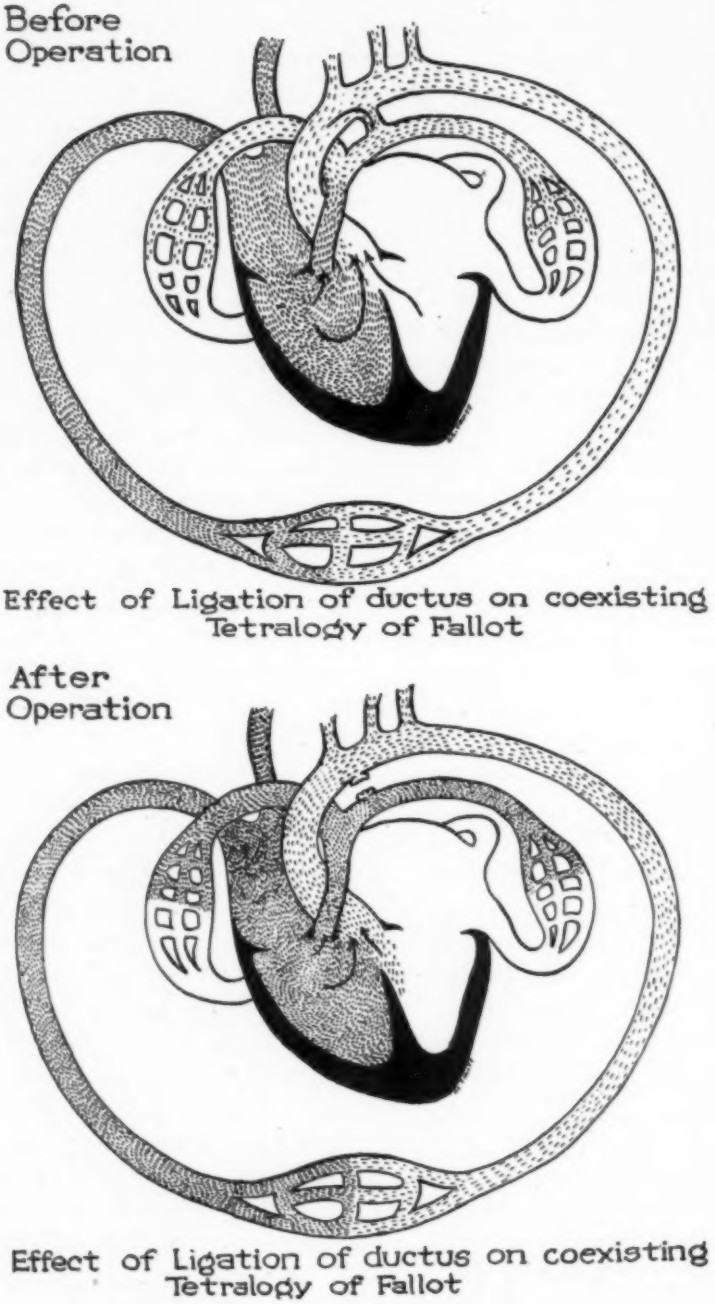


FIGURE 4: These schematic diagrams illustrate the increased cyanosis that results from ligating or dividing a patent ductus arteriosus in the presence of a coexisting pulmonary artery stenosis. In reverse order they indicate how the cyanosis of Tetralogy of Fallot may be relieved by establishing an artificial ductus arteriosus (Blalock-Taussig operation).

dence of a particularly large shunt. Since Touroff[83,40] demonstrated that the peripheral blood stream may be sterilized by ligation of a patent ductus arteriosus complicated by bacterial pulmonary endarteritis, the presence of such superimposed infection is generally accepted as an urgent indication for surgical intervention.

Whether or not surgery is indicated as a purely prophylactic procedure has been open to some debate. In skilled hands the surgical mortality has been so low, even during the developmental era of this procedure, that the hazards of surgery seem well justified when there is prospect of adding some twenty or thirty years of life to a child's expectancy. The fact that an occasional duct has appeared to obliterate spontaneously even during the later years of life[10,35] provides some basis for argument against prophylactic surgery. This, however, does not alter the fact that the case who lives out his normal life span is the exception in the group as a whole.

I would like to emphasize again that the presence of other cardiac anomalies, for which the ductus acts as a compensatory blood channel, provides a definite contraindication to interruption of this vessel. I understand that such surgery has been performed and of course with deleterious results (Fig. 4). In general it may be stated that persistent cyanosis per se is a contraindication to ligation or division of a patent ductus arteriosus.

The technics that have been used are well presented by Gross[12,17] and have been modified very little by others. Inhalation anesthesia through a closed system with an endotracheal tube is preferred. The surgical approach is through the third intercostal space anteriorly, or through the periosteal bed of the fourth or fifth resected rib posteriorly as advoated by Harrington.[20] The diagnosis is confirmed by palpating the duct and the thrill which is transmitted to the pulmonary artery. The phrenic and vagus nerves are identified and the mediastinal pleura is incised between them. The ductus arteriosus is isolated by blunt dissection avoiding injury to the vagus and recurrent laryngeal nerves and to the extension of pericardium that sometimes approaches the duct from the pulmonary artery. Before the duct is permanently interrupted it should be occluded by compression. Should cyanosis appear during this maneuver the duct must be left intact since there is probably an associated pulmonary stenosis. On occlusion of the duct the palpable thrill will immediately disappear or markedly diminish. Some thrill may persist if the pulmonary artery is greatly dilated.

Simple ligation of the duct with a single strand of non-absorbable suture material, as formerly practiced, has been found unsuitable because in too many instances the shunt became

FIGURE 5: (1) Patent ductus arteriosus exposed. Flap of mediastinal pleura and phrenic nerve retracted with silk traction sutures. (2) Tape ligatures at each end of ductus arteriosus. Introduction of transfixion suture. (3) Ductus arteriosus obliterated with two tape ligatures and transfixion suture.

re-established by recanalization around the ligature. To avoid such operative failures other techniques have been devised: multiple ligatures; wrapping the ligated duct with a type of cellophane which causes a severe sclerosing reaction;[19] obliteration of the entire length of the duct, including, perhaps, portions of the adjoining arterial walls, with tape ligatures; the use of mattress or transfixion sutures in conjunction with the above methods; and finally division of the duct between clamps with suture closure of its divided ends by the technic which Gross[17] has described and used with such spectacular success. An additional technic for dividing the duct has been used by Crafoord[8] who recommends clamping the aorta while excising the aortic end of the duct.

The technic to be used must depend largely on the experience and skill of the operator. In discussing the ligation of large arteries in continuity, Reid[33] has shown that the size of the ligating material should increase in direct proportion to the size of the artery to be ligated. A fine ligature, allowing less time for repair of the devitalized segment, may cut through the wall of the vessel with the result that hemorrhage or re-establishment of the lumen occurs. It is also important that a ligature be tied in such a manner as to obliterate the lumen of the vessel without rupturing its media and intima. Careful obliteration of the ductus arteriosus with two or more ligatures of umbilical tape, reinforced perhaps with a mattress or transfixion suture, has appeared to be adequate in most instances (Figs. 5 and 6). I have had no experience with the use of cellophane. Since the reaction from this material can be so severe as to cause obliteration of the aorta in experimental animals[31] I would not consider using reactive cellophane for a ductus arteriosus until it has been adequately demonstrated that it will never cause injury to the adjacent nerves, great vessels and lung tissues. Such risk is not justified when other suitable methods are available.

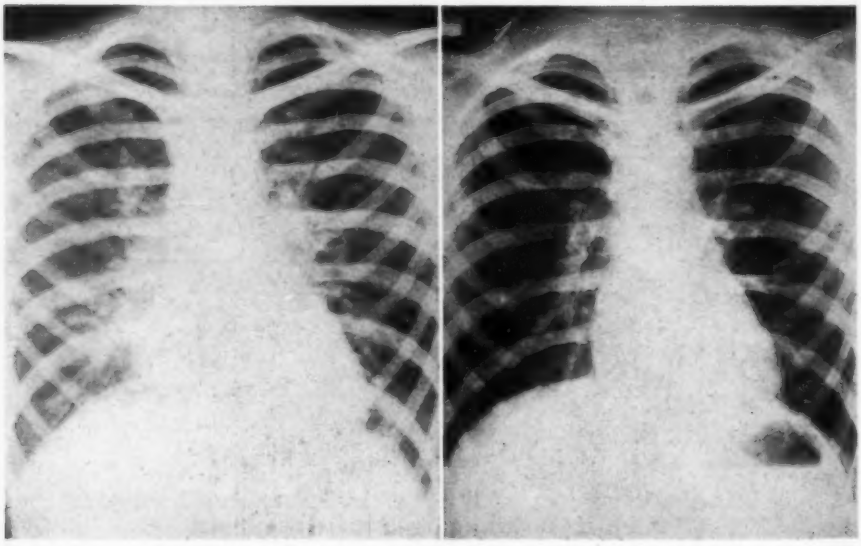
As my experience with vascular surgery has increased I have become more and more convinced that division of a patent ductus arteriosus is preferable to its ligation, as long as there is no increased hazard to the patient. While the technic of Gross has been used with remarkable success by him, by Jones and by other surgeons, it is still true that an occasional death from hemorrhage has resulted. There is some danger that the clamps may slip or become dislodged after the duct has been divided and before the divided ends have been sutured. The first time I divided a ductus arteriosus its divided ends retracted between the jaws of the clamps. The profuse and terrifying hemorrhage which ensued was controlled with greatest difficulty. While this accident may be considered a reflection on my technic, others

have had the same experience (sometimes with a less favorable outcome) and it is one which I would not care to duplicate. It has also been observed that for exceptionally short ducts the Gross technic is not applicable.

Crafoord, by completely clamping the aorta, largely eliminates the danger of serious hemorrhage. But clamping the aorta of normal dogs for even short periods (ten to fifteen minutes) has frequently resulted in paralysis of their posterior extremities.[2,18] One must anticipate the possibility that this same serious complication may occur in humans who do not have the increased collateral circulation such as is associated with coarctation of the aorta.

In November 1946 Potts, Smith and Gibson[32] described a clamp which they had devised in order to isolate a segment of the aorta while making a side-to-side anastomosis of the aorta to the pulmonary artery. This clamp permits some blood to flow through the aorta, around the compressed portion of its wall that is to be incised. It occurred to me that the same clamp would be applicable to the reverse procedure, division of a patent ductus arteriosus, and I have recently had occasion to use it in such an operation (Fig. 7).

A five year old girl was operated on April 18, 1947. A posterolateral approach was used. After determining that an uncomplicated patent ductus arteriosus was present the segment of the



(1) (2)

FIGURE 6 (Case No. 6): M.D. (1) PA chest film, 12-1-45; prominent pulmonary artery segment with increased vascular shadows extending into the lung fields. (2) PA chest film, 1-4-47; following ligation of patent ductus arteriosus.

aorta opposite the ductus was isolated, dividing one intercostal artery. The aortic clamp was then applied compressing the inferior wall of the aortic arch and interrupting the flow of blood through the ductus arteriosus, as evidenced by immediate disappearance of the thrill in the pulmonary artery. Isolation of the ductus, which was large both in diameter and in length, then proceeded with remarkable ease. Following novocaine infiltration, the vagus nerve was retracted gently with tape in order that it and the recurrent laryngeal nerve would not be traumatized. The pulmonary artery end of the duct was then doubly ligated and transfixed and the duct was divided between the ligatures and the clamp. The aortic end of the duct was sutured with a running mattress stitch of No. 00000 Deknatel which approximated its walls, intima to intima. This was followed by a running glover's stitch. On slowly releasing the clamp a little oozing occurred near the middle of the suture line and an interrupted mattress suture was placed at this site before the clamp was finally removed. No further

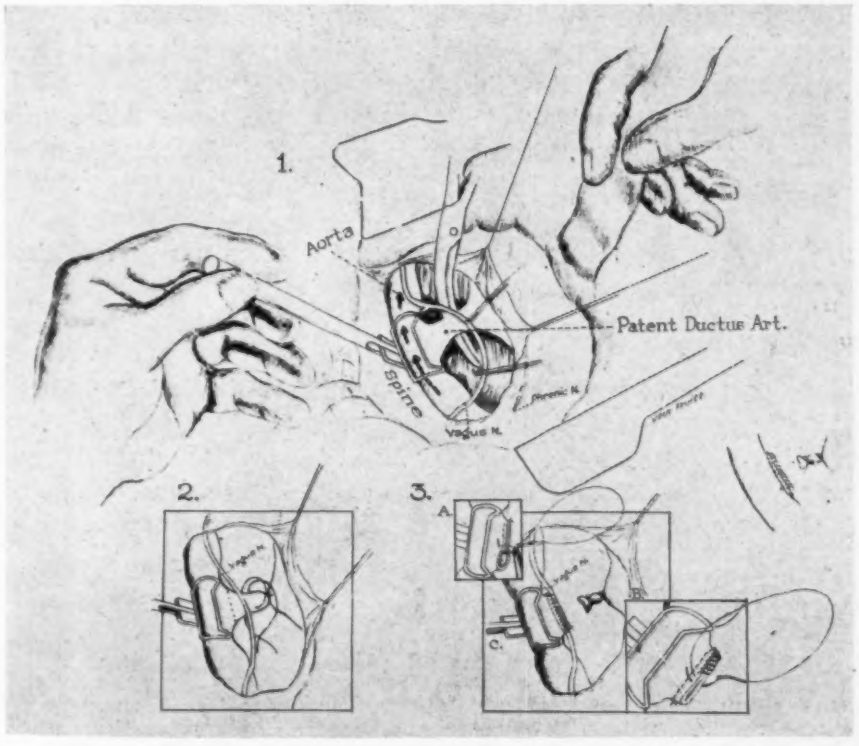


FIGURE 7: (1) Aortic clamp (Potts-Smith-Gibson) in place, isolating aortic end of patent ductus arteriosus. (2) Following ligation of pulmonic end of ductus a transfixion suture is being placed. Dotted line indicates site at which ductus will be divided. (3) Suturing of aortic end of duct. A - running mattress suture. B - running glover's stitch. C - completed suture with aortic clamp still in place. Some aortic circulation has been maintained. Insert: Clamp removed.

bleeding occurred. The pleura was reapproximated over the divided duct and the vagus nerve with a few interrupted chromic stitches and the chest wall was closed re-expanding the lung with catheter suction. Postoperatively the child had some hypertension but following a small venisection the blood pressure stabilized at about 120/80. Preoperatively the blood pressure was 110/0. Postoperative convalescence was smooth. No thoracenteses were required and there was no leg paralysis.

It is my impression that this technic offers the following advantages: 1) The danger of serious hemorrhage from the aorta is eliminated. 2) Isolation of the entire duct is relatively easy and rapid because of wider dissection and exposure with diminished danger from hemorrhage. 3) The vagus nerve is well exposed and can be retracted in such a manner that the recurrent laryngeal nerve is less likely to be injured during isolation of the duct. 4) The aortic end of the duct can be divided and sutured close to the aorta, or actually excised, so that no pouch or aneurysmal dilatation of the wall of the aorta will exist. 5) Clamps are not placed close to the suture line where interference with blood supply might promote necrosis and sloughing of the sutures. 6) This technic would probably be applicable to very short ducts or those with no length at all. If necessary the pulmonary artery could be temporarily occluded, proximal and distal to the duct, while the duct is divided and while its pulmonary end is sutured in the same manner as its aortic end. I propose this technic as one that is safe and more generally applicable than others that have been used.*

Complications

The operative and postoperative complications that have been or may be encountered include the following:

1. Hemorrhage.
2. Shock.
3. Vago-vagal reflexes.
4. Cardiac arrest.
5. Myocardial infarction.
6. Hypertension (transient).

*A recent experience demands that I add a footnote in regard to this technic. A ductus arteriosus was divided using the largest available Potts clamp. The clamp was not large enough to isolate completely the aortic end of the ductus since the aorta was exceptionally large. A strip of umbilical tape was used to compress the non-occluded segment of the aorta against the clamp at its proximal end. This ill-advised maneuver resulted in a tear in the aortic wall which progressed proximally and resulted in uncontrollable hemorrhage.

Division of three additional ducti using the Potts clamp have confirmed the advantages of the technic. But the necessity for having a clamp of suitable size before proceeding with this type of operation must be emphasized.

7. Left recurrent laryngeal nerve paralysis.
8. Phrenic nerve paralysis.
9. Recanalization of the duct.
10. Rupture of the duct or of an associated aneurysm.
11. Aorto-bronchial fistula.
12. Death from associated cardiac anomalies.
13. Tension pneumothorax.
14. Pleural effusion, hematothorax and fibrothorax.
15. Empyema.
16. Pulmonary embolism and infarction.
17. Pulmonary atelectasis.
18. Pneumonia.
19. Bacterial endarteritis and septicemia.
20. Keloid scar.
21. Ligation of wrong vessel.

John C. Jones has presented an excellent review of complications encountered in the surgical treatment of sixty-one consecutive cases of patent ductus arteriosus,[22] including fifty-three ligations and eight divisions. Since this report he has operated, by division, nine additional cases.[23] The series includes no cases in which bacterial endarteritis was known to exist prior to surgery. In the entire group (70 cases) there were two deaths, one surgical death from hemorrhage and cardiac arrest (1.4 per cent) and one late death from staphylococcus aureus endocarditis. In two cases there were large pulmonary hemorrhages several months postoperatively. In one of these an aorto-bronchial fistula was found and surgically repaired. In the other, presumed to have the same complication, spontaneous healing seems to be in progress. Ten of the fifty-three that were ligated have had more or less recurrence of the typical murmur, due either to incomplete occlusion of the vessel or to recanalization around the ligatures. Postoperative pulmonary atelectasis occurred in five cases, all responding to treatment. Empyema occurred twice, following pneumonia in one. In three cases there was recurrent laryngeal nerve involvement, transient in one, persistent (but with considerable voice improvement) in two.

Results

The results of surgical treatment of persistent patent ductus arteriosus and of the various technics employed cannot be finally evaluated for many years. It will take a half a century to determine incontrovertibly that the children subjected to surgery during these past eight years have had their life expectancy materially prolonged and that the surgical mortality, however small, has been amply justified by the promise of longer life and better health for those in whom the surgery is considered to have been successful.

Shapiro[36] has recently collected information on 643 surgically treated cases reported by forty-six surgeons. In seventeen of these no patent ductus arteriosus was found. A patent ductus arteriosus was ligated in 343 uncomplicated cases with a surgical mortality of 4.9 per cent and recanalization in 8.7 per cent. In eighty-eight cases complicated by subacute bacterial endarteritis surgical ligation resulted in a mortality of 28.4 per cent and recanalization in 4.5 per cent. Division of the ductus arteriosus was carried out in 182 uncomplicated cases with only one death and in thirteen infected cases with two deaths, one of which resulted from continued infection following successful section.

While one may suppose that published reports tend to reflect only the more favorable results and that early mishaps or failures tend to discourage the surgeon from further attempts and from recording his experiences, we may be encouraged by the realization that improvements and standardization in technics tend to lower the risk of any new surgical procedure. Particularly encouraging is the knowledge that surgeons, like Gross and Jones, who have had the widest experience in this field, have maintained a consistently low mortality, well below the average. It may be assumed that a growing number of surgeons, benefiting by their experience and training, will do the same.

In cases complicated by bacterial endarteritis and endocarditis the surgical mortality tends to be very much higher. It was in this group, however, that almost a 100 per cent mortality had formerly prevailed. As mentioned above, sterilization of the blood stream by antibiotic and other medical therapy does not protect against reinfection nor eliminate heart strain and the irreversible changes that occur. The indication for surgery in the presence of this complication continues to remain more urgent than in others. It is particularly heartening that an early surgical mortality of 50 per cent[35] in this group appears now to have been reduced to 28.4 per cent.[36] Medical therapy, of course, should be used in conjunction with surgery.

All surgeons, cardiologists and pediatricians seem to have been favorably impressed by the changes that follow interruption of a persistent patent ductus arteriosus. The usual postoperative course is smooth. Children who previously were poorly developed will ordinarily show a rapid and marked gain in weight; irritability and fatigability diminish, and there is a gradual improvement in physical development, vigor, endurance and vitality. Any or all of the symptoms previously enumerated will tend to disappear.

My own experiences are summarized in Chart II and I shall present in greater detail a few of the cases in which conditions or complications may prove of particular interest.

B. O., a twenty year old college student, was referred for treatment in May 1944, by Dr. C. P. Wilson, a diagnosis of uncomplicated patent ductus arteriosus having been made.

A left thoracotomy was performed on June 8, 1944 under cyclopropane anesthesia administered through an intratracheal tube. A very large, short duct was isolated without mishap. An attempt was made to divide the duct in the manner described by Gross,[17] but following division of the duct its divided ends retracted between the jaws of the outermost clamps in such a manner that no cuff of tissue was available for suture. In attempting to place an additional clamp proximal to the aortic end of the duct the remaining clamp came off completely leaving a wide open hole in the aorta, with a tremendous gush of blood shooting several feet into the air. The bleeding was controlled by introducing a thumb into the aorta. The vessel edges were finally re-approximated with the aid of heavy hemostats and closure was carefully completed with an interlacing suture. In this emergency it was impossible to avoid some injury to the recurrent laryngeal nerve. Similar difficulties were encountered with the pulmonary end of the duct. However, here the closure was accomplished more easily and with relatively little blood loss. During the operation 1,500 cc. of blood were transfused. The immediate postoperative course was smooth. The patient was discharged from the hospital on the sixteenth postoperative day. Four days after discharge he was readmitted to the hospital complaining of fever, chest pain, dry hacking cough, and slight hemoptysis. The findings were compatible with a diagnosis of pulmonary embolism. Thoracentesis yielded 450 cc. of serosanguineous fluid. No organisms could be cultured from this fluid. At the time of thoracentesis 40,000 units of Penicillin were introduced into the pleural space and parenteral Penicillin therapy was started. There was gradual improvement in the patient's symptoms, his temperature and pulse returned to normal. On the fourteenth day of the second admission he was discharged home. He remained on bedrest for one week and then started on graduated uptime. He gained weight and gradually resumed normal activity. For several months after operation he remained hoarse but the quality of his voice slowly returned to normal. Five months after operation his blood pressure was 136/82. A soft systolic murmur to the left of the sternum persisted. Fluoroscopic examination showed slight fullness of the shadow of the pulmonary artery. When last seen, three years following surgery, he was working in Hollywood, leading a very active life which included sports such as swimming and basketball.

Comment: Despite previous testing of the clamps, the elastic bands placed about the clamp handles did not provide sufficient tension to prevent retraction of the vessel walls and dislodgement of the clamps. Such a technical failure can usually be avoided. However, I would advocate some modification of the technic which would completely obviate this hazard. I believe that the clamp described by Potts et al,[32] placed on the aorta opposite the aortic end of the duct, eliminates the danger of serious hemorrhage while the duct is being divided and while its divided ends are being sutured.

The source of the postoperative pulmonary embolism was not

CHART II

Case No.	Sex	Age at opn.	History	Physical Findings	BP Preop.	BP Postop.	EKG	Operation	Postoperative Course	Follow-up
1. P.M.	F.	9	(1) Murmur at 4 mos. (2) Fatigue. (3) Nosebleeds. (4) Weight loss.	Machinery murmur second left interspace, accentuated P_2, prominent conus Corrigan pulse.	124/60	130/85	Sinus arrhythmia normal axis.	Anterior approach, ductus ligated in continuity with umbilical tape at each end.	Venesection on 3rd postoperative day reduced BP 150/100 to 130/70.	Relief of fatigability and irritability. No nosebleeds for 1 year. Increased activity.
2. B.O.	M.	20	(1) Heart murmur since birth. (2) Inability to gain weight. (3) Slight fatigability.	Machinery murmur, right ventricular enlargement and hilar dance to fluoroscopy.	115/55	136/82	Left axis deviation.	Anterior approach, duct divided by Gross technique, serious hemorrhage from each end, injury to recurrent laryngeal nerve.	On 20th postoperative day patient had attack resembling pulmonary embolism.	Relief of fatigue. Weight gain. Persistent systolic murmur. Hoarseness, gradually cleared.
3. C.S.	F.	3	(1) Transient cyanosis with crying. (2) Fainting spells. (3) Fever.	Machinery murmur and thrill, heart enlarged, capillary pulse, one of 4 blood cultures showed Staph. aureus (contaminant?).	136/66	130/80	Normal axis.	Anterior approach, duct ligated in continuity with umbilical tape at each end.	Smooth. Thoracentesis on 3rd postoperative day.	Relief of fainting spells, increased exercise tolerance.
4. J.F.	M.	25	(1) Blue @ birth. (2) Fatigability. (3) Pain in left hemithorax. (4) Palpitation. (5) Narcoleptic attacks.	Continuous murmur, no thrill, capillary pulse, Corrigan pulse.	130/94	120/84	Left axis deviation, negative T_2.	Anterior approach, duct 2 cm. diameter ligated with umbilical tape at each end in continuity.	Uncomplicated. Thoracentesis (4th postoperative day) yielded 400 cc.	Relief of "narcolepsy," increased exercise tolerance.
5. J.W.	M.	5	(1) Blue spell @ 2 mos. (2) Fatigue. (3) Dyspnea. (4) Inability to speak clearly.	Continuous murmur and thrill in 2nd left interspace, questionable apical systolic murmur, pulmonary segment elongated & bulging to fluoroscopy.	95/40	95/40	Normal axis.	Anterior approach. Non-patent **ligamentum arteriosum**, narrowing of ascending aorta. Probable aortic septum defect. Exploration only.	Uncomplicated.	Repeated respiratory infections. Poor progress in school. Impaired speech.
6. M.D.	F.	10	(1) Fatigability. (2) Irritability. (3) Recurrent respiratory infections. (4) Eczema—food allergies.	Machinery murmur and thrill in 2nd left interspace, widened pulse pressure.	120/0	106/75	Normal axis.	Anterior approach, short, wide ductus ligated in continuity with umbilical tape at each end.	Uncomplicated.	Relief of fatigability and irritability. Still repeated upper respiratory infections.
7. J.S.	F.	19	(1) Murmur @ 1 yr. (2) Palpitation. (3) Precordial pain on exertion.	Machinery murmur and thrill in 2nd left interspace, capillary pulse, right	110/68	122/80	Normal axis.	Anterior approach, duct 2 cm. in diameter ligated in continuity	Uncomplicated.	Incisional keloid. Dyspnea and palpitation

			ratory infections. (4) Eczema—food allergies.	widened pulse pressure.				at each end.		respiratory infections.
7. J.S.	F.	18	(1) Murmur @ 1 yr. (2) Palpitation. (3) Precordial pain on exertion. (4) Dyspnea. (5) Ankle edema.	Machinery murmur and thrill in 2nd left interspace, capillary pulse, right ventricular outflow tract and pulmonary artery enlarged.	110/68	122/80	Normal axis.	Anterior approach, duct 2 cm. in diameter ligated in continuity with umbilical tapes at each end. Marked dilation of pulmonary artery over which a thrill was still palpable after the duct had been securely obliterated.	Uncomplicated.	Incisional keloid. Dyspnea and palpitation relieved. Persistent systolic murmur over 2nd left interspace.
8. F.S.	F.	9	(1) Murmur @ 9 yrs.	Machinery murmur, 2nd left interspace, capillary pulse, exaggerated hilar lung markings.	108/50	120/80	Normal axis.	Anterior approach, ductus ligated with heavy silk and divided after hemorrhage from posterior wall. Aorta clamped 35 minutes during division.	Uncomplicated. EKG changes interpreted as "myocardical infarction" in vector precordial leads.	Well, no evidence of paralysis, EKG reverted to normal.
9. D.F.	F.	2	(1) Low vitality. (2) Dangerously ill with recurrent respiratory infections. (3) Failure to grow normally.	Systolic murmur and thrill over 2nd and 3rd left interspaces. No diastolic component. Marked enlargement of heart.	116/26	——	Normal axis.	Patient expired due to cardiac arrest during isolation of ductus.	Autopsy: atelectasis of both lungs. Patent ductus arteriosus sole cardiovascular anomaly. All chambers of heart enlarged.	
10. J.B.	M.	22	(1) Murmur found at Army examination. (2) Fever. (3) Pain in left side of chest. (4) Loss of weight and strength. (5) Dyspnea.	Continuous murmur in 2nd and 3rd left interspaces, apical systolic murmur. Dullness and diminished breath tones over left hemithorax.	130/100	124/96		Posterolateral approach. Fibrinous deposition on left lung decorticated. Long ductus ligated in continuity with umbilical tape.	Uncomplicated. Drainage tubes removed on second postoperative day.	Improved weight, strength and pulmonary function.
11. D.C.	M.	9	(1) Spells of cyanosis during first 2 mos. of life. (2) Easy fatigability.	Systolic and early diastolic murmur in 2nd and 3rd left interspaces.	120/70	116/80	Right axis deviation.	Anterior approach, ductus ligated in continuity with umbilical tape at each end.	Uncomplicated.	
12. P.N.	F.	5	(1) Murmur present since birth. (2) Subnormal physical development.	Continuous murmur in 2nd and 3rd left interspaces. Palpable thrill. Corrigan pulse.	110/0	120/80	Right axis deviation.	Posterolateral approach, ductus divided using Potts-Smith-Gibson clamp on aorta.	Uncomplicated.	

definitely established. There was no evidence of phlebothrombosis. It is possible that trauma, contingent on the operative difficulties, favored the formation of a thrombus at the pulmonic end of the divided ductus and that this thrombus later became dislodged. The fact that only the left lung was involved is compatible with this explanation. A pre-existing bacterial endarteritis had not been diagnosed or suspected.

J. F., a twenty-five year old fireman, was referred for treatment in June 1944, by Dr. Homer Rush. The patient complained of attacks of listlessness since the age of fourteen. Typically the attacks occurred daily at about 10:00 A.M. and 2:00 P.M. Occasionally the patient would have difficulty remaining awake while driving a car and at other times. He also noted occasional attacks of momentary mental confusion. He had been told that he had narcolepsy and benzedrine, ten milligrams two or three times daily, had partially controlled the symptoms. His mother stated that he was a blue baby at birth and that cyanosis persisted for the first ten days of life. There had been no cyanosis since that time. Slightly increased fatigability was noticed and he complained of an ill-defined pain in the left upper part of his chest. There was occasional slight faintness but no actual loss of consciousness. Although the diagnosis of patent ductus was not clearcut the patient repeatedly insisted that exploration be performed to determine whether or not a correctable defect were present.

On April 17, 1945 a patent ductus arteriosus, measuring two centimeters in diameter, was ligated with two widths of three-eight inch umbilical tape. The postoperative course was smooth. Since surgery the patient has been completely relieved of his previous symptoms. His work in a new occupation as a medical illustrator shows considerable aptitude and promise.

Comment: This patient had read a good deal of medical literature concerning patent ductus arteriosus and he was convinced that obliteration of a shunt would cure his "narcolepsy." His previous symptoms had prevented him from undertaking anything which required sustained effort or concentration. He was handicapped considerably in his University studies, in his choice of occupation and in pursuance of normal activities. It may be surmised that cerebro-vascular circulation may improve following elimination of a ductal shunt and that this accounts, at least in part, for the gratifying result obtained in this case.

F. S., a nine year old girl was referred by Dr. Roderick Norton of Tacoma, Washington. A diagnosis of uncomplicated patent ductus arteriosus had been made.

Operation was performed on September 2, 1946 through an incision in the second left interspace. There was a large, short ductus arteriosus and on exposing its presenting surface it was immediately noted that the duct had an extraordinarily thin wall which bulged outward when supporting structures had been removed. Because of this finding extreme caution was observed. Nonetheless, when the duct had been partially

dissected, there was a sudden profuse hemorrhage from its posterior aspect. The duct being extremely short it seemed unduly hazardous to proceed immediately with its isolation since further tearing of its wall and dangerous blood loss were likely to ensue. While bleeding was controlled with digital pressure the aorta was isolated and occluded, proximal to the duct, with a large hemostat. Dissection was then continued until the duct was isolated. After both ends of the duct had been ligated and transfixed the vessel was divided. The aortic clamp was then released producing a transitory fall in the blood pressure which was not marked. Further bleeding from the aortic end of the duct required reapplication of the aortic clamp while supporting sutures were introduced. The aorta was occluded for a total of thirty-five minutes. Postoperative mucus accumulation in the tracheobronchial tree required removal by suction. An electrocardiogram taken seven days after the operation showed changes consistent with left ventricular ischemia. The patient had no complaints referrable to the heart following operation and electrocardiographic changes have since reverted to normal.

Comment: As reported by Blalock and Park[2] and by Gross and Hufnagel[18] prolonged occlusion of the aorta in dogs has often resulted in paralysis of the posterior extremities. In this case the aorta was clamped for a period of twenty minutes and again for a period of fifteen minutes. Fortunately no evidence of any paralysis resulted. I understand that Crafoord clamps the aorta routinely when dividing a patent ductus arteriosus and apparently he has had no complications when employing this technic. Use of the clamp described by Potts, Smith and Gibson[32] should provide additional safety in a procedure of this type. We have been most interested in the temporary electrocardiographic changes that occurred in this case and have not, as yet, arrived at a satisfactory explanation for these. They may have resulted from a transient coronary insufficiency resulting from a fall in blood pressure when the aortic clamp was removed.

D. F., aged two and one half years was referred by Dr. Frank Maddison of Tacoma, Washington. She was admitted to Doernbecher Hospital because of slow growth and severe respiratory infections. She had had two severe attacks of respiratory infection with fever, nasal obstruction, bronchitis and rapid breathing. During these attacks the child was very ill, her temperature rising as high as 106 rectally. According to her family physician on each occasion she had "almost succumbed." The child often became very pale and white upon over-exertion but there was never any definite history of cyanosis. She had been seen by Dr. Helen Taussig who had confirmed a previous diagnosis of patent ductus arteriosus and had advised early surgery. Electrocardiographic and fluoroscopic findings indicated that all four chambers of the heart were markedly enlarged. In view of the enlarged, dilated heart and history of severe dyspnea, the patient was digitalized before operation.

Thoracotomy was carried out through incision in the second left anterior interspace. The pulmonary artery was markedly dilated. There was a continuous machinery thrill felt over the pulmonary artery and aortic

arch. A patent ductus arteriosus was partially isolated. About one hour after the induction of anesthesia the instruments were removed from the chest so that the lung could be re-inflated. Before the lung could be expanded, it was noticed that the heart had stopped completely and suddenly. Slight irregular systoles involving the whole heart could be palpated, but there was no demonstrable peripheral pulse or blood pressure. Cardiac massage and bag breathing were started. Fifty-five minutes after cardiac massage had been instituted further attempts at resuscitation were abandoned.

Comment: In preliminary correspondence with the child's family physician I had urged that every attempt be made to carry her along without surgery for at least another year or two. Having followed her closely from birth, he stated that "on two occasions she contracted a mild upper respiratory infection and almost succumbed each time," and he was convinced that surgery would have to be performed soon, if at all. Dr. Taussig had apparently concurred in this opinion.

The cause for sudden cardiac arrest is still obscure. In view of the systoles involving the whole heart, it is improbable that the failure was on the basis of ventricular fibrillation. The possibility of complete sino-auricular block should be considered. Autopsy showed massive bilateral pulmonary atelectasis, hyperemia and edema of the lungs, petechial hemorrhages into the pleura and a patent ductus arteriosus approximately 12 mm. in diameter which was the only congenital cardiac defect. There was marked dilatation and hypertrophy of all chambers of the heart.

J. B., a twenty-two year old truck driver, was referred for treatment by Dr. R. E. Herron in August 1946. He complained of pain in the left side of the chest posteriorly, loss of weight and strength and slight morning cough, which was not productive. He first learned that he had a heart murmur when he was rejected for army service. In June 1946 he had had an acute attack of fatigue, chills, fever and loss of appetite. The day following onset of this illness he noticed pain in the left lower part of his chest. He was admitted to a local hospital where he was told that he had "pneumonia." He required oxygen for the first two days. Penicillin was given for four weeks. Nine days after the onset of his illness a left pleural effusion was found. This was aspirated numerous times. His temperature gradually returned to normal. He was discharged after three weeks hospitalization. He gained weight and strength but continued to have a slight non-productive morning cough and dull constant pain in the left lower part of his chest posteriorly. The pain was worse when the patient was fatigued. He was admitted to my service six weeks after the onset of his febrile illness.

Examination showed a tall, thin white male. There was limited respiratory excursion of the left hemithorax and flatness to percussion in the left lower lung field. Breath tones were weak and crepitant rales were heard occasionally in this area. Pulse rate was 128. A continuous machinery-like murmur was heard in the second and third left interspaces. No thrill was felt. Following admission to the hospital he had

recurrent attacks of vomiting and fever. Thoracenteses yielded relatively small amounts of thin bloody fluid. The fluid showed no organisms on smear and culture. Cultures for tubercle bacilli were negative. A diagnosis of tuberculous pleurisy with effusion was considered until intradermal tuberculin tests were found to be negative, even in concentrated dosage. He gained weight on bedrest and following a final febrile episode of short duration he was discharged home. He was allowed to increase his activities gradually and remained asymptomatic. However a marked contracture and fixation of the left hemithorax persisted. The lower portion of his left lung field remained radio-opaque, flat to percussion and breath sounds could not be heard in this area.

Six months after discharge he was readmitted to the hospital. On January 16, 1947 under endotracheally induced cyclopropane anesthesia, a thoracotomy was performed through the bed of the resected fourth rib using a posterolateral approach. The left lung was enveloped by a thick and very dense fibrinous membrane measuring two or more centimeters in thickness. There was no free fluid.

The lung was completely freed from the chest wall and then, carefully, the visceral portion of the closely adherent fibrotic membrane was removed. The surface of the lung under the membrane appeared dark and airless but with slight positive pressure, applied by the anesthetist, good aeration and re-expansion was gradually obtained. The more accessible areas of the parietal portion of the membrane were then removed with the hope that increased mobilization of the rib cage might result. The diaphragm was elevated and fixed, the costophrenic angle being completely obliterated with organized fibrin. This area was practically inaccessible from the site of exposure and little attempt was made to free the edges of the diaphragm.

A ductus arteriosus was found and isolated. It had considerable length but was only about one centimeter in diameter. Dissection around the duct was rather difficult because of the marked fibrous tissue reaction that extended deep into the mediastinum. When both ends of the duct were obliterated with ⅜ inch umbilical tape ligatures, the thrill, previously felt over the pulmonary artery, disappeared immediately. Additional measures seemed unnecessary and the wound was closed while lung expansion was maintained, two intercostal tubes, anterior and posterior, being left in place for postoperative suction drainage. These tubes were removed on the third postoperative day when breathing exercises were instituted.

Convalescence was entirely uneventful and the patient returned in about six weeks to his former occupation, i.e. truck driving.

Comment: This patient's febrile illness, pleural exudate and fibrothorax have never been adequately explained. Pathological examination of the membrane showed nothing to suggest a tuberculous etiology and tuberculin tests had been repeatedly negative. One possibility is that he had a bacterial endarteritis complicating his patent ductus arteriosus, that the blood stream infection had responded to the Penicillin given elsewhere, and that the episodes recurring after we saw him represented pulmonary embolism from breaking off of residual vegetative material in the pulmonary artery.

DISCUSSION

Unequivocably it may be stated that the surgical treatment of patent ductus arteriosus is indicated under certain conditions: 1) in the presence of retarded development, 2) when evidence of heart strain occurs, 3) in the presence of a complicating subacute bacterial endarteritis.

In addition, the concensus appears to be that all children and young adults, who have an uncomplicated patent ductus arteriosus the spontaneous obliteration of which is unlikely to occur, are candidates for surgical treatment in order that the hazards of this condition may be prevented and the survival expectancy prolonged. Elective surgery in these cases should be postponed until after four years of age and is most readily performed between the ages of five and ten.

The surgical mortality in the series collected by Shapiro was about 5 per cent, and varies indirectly with the skill and experience of the surgeon. In cases complicated by bacterial endarteritis, the surgical mortality is still high (28.4 per cent). Despite the fact that chemotherapeutic and antibiotic agents have been of value in the treatment of bacterial infection of the blood stream, even this mortality offers less hazard to the patient than failure to eliminate the abnormal shunt.

SUMMARY

The literature concerning patent ductus arteriosus is briefly reviewed.

A persistent patent ductus arteriosus may be eliminated by surgical division or ligation. When the duct remains open after the first year of life, spontaneous obliteration is not likely to occur. Unless associated with other cardiovascular anomalies, its surgical treatment in children over the age of four years should be considered in order that life expectancy may be increased. The surgical mortality in skilled hands is sufficiently low to justify this conclusion.

Since recanalization has frequently followed ligation of a patent ductus arteriosus, division is considered the procedure of choice. A modified technic for division of a patent ductus arteriosus is presented. This technic is considered to have several advantages, being safer and more widely applicable than the technics now in use.

Twelve cases are reported in which the diagnosis of patent ductus arteriosus was made and operation performed. In one case the preoperative diagnosis of patent ductus was in error. There was one surgical (anesthetic?) death.

Other cases reported in detail include: one in which massive hemorrhage occurred following division of the duct and in which convalescence was complicated by the occurrence of pulmonary emboli; one in which the aorta was clamped for a total of thirty-five minutes while hemorrhage was being controlled and the duct divided; one case in which attacks resembling narcolepsy were apparently relieved following ligation of the duct; and one in which decortication of the lung was performed at the time of ductus ligation.

I am indebted to Dr. Elton Watkins, Jr. for assistance in reviewing the cases herein reported and to Mr. Jack Fruitt for the illustrative drawings.

RESUMEN

Se ha revisado brevemente la literatura relativa al conducto arterial persistente.

Puede eliminarse un conducto arterial persistente por medio de la división o la ligadura quirúrgicas. Cuando el conducto persiste después del primer año de vida, es improbable que ocurra la obliteración espontánea. A menos que esté asociado con otras anomalías cardiovasculares, debe considerarse el tratamiento quirúrgico en niños de más de cuatro años a fin de poder prolongar sus expectativas de vida. La mortalidad quirúrgica en manos expertas es lo suficientemente baja para justificar esta conclusión.

Ya que la recanalización ha seguido frecuentemente a la ligadura del conducto arterial persistente, se considera que la división es el procedimiento preferible. Se presenta una técnica modificada para la división del conducto arterial persistente. Se considera que esta técnica ofrece varias ventajas, presenta menos peligros y es de aplicación más extensa que las técnicas ahora empleadas.

Se informa sobre doce casos en los que se hizo el diagnóstico de conducto arterial persistente y se ejecutó la operación. En un caso el diagnóstico preoperatorio de conducto persistente fue erróneo. Hubo una muerte quirúrgica (anestésica?).

Otros casos sobre los cuales se informó en detalle incluyen: uno en el que ocurrieron hemorragias masivas subsiguientes a la división del conducto y en el que embolias pulmonares complicaron la convalescencia; uno en el que se pinzó la aorta por un período total de treinta y cinco minutos mientras se dominaba la hemorragia y se seccionaba el conducto; un caso en el que la ligadura del conducto aparentemente alivió ataques parecidos a narcolepsia y uno en el que se llevó a cabo el descortezamiento del pulmón al mismo tiempo de la ligadura del conducto.

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Discussion

JOHN ROBERTS PHILLIPS, M.D., F.C.C.P.
Houston, Texas

Although it is felt that division and suture of the ends of the ductus will give the best anatomical results, there are cases in which the ductus is too short to carry out such a procedure and there is always the hazard of severe uncontrollable hemorrhage should one of the clamps be dislodged. Consequently, it is advisable

to ligate the ductus by "purse string" suture and mattress stitches as proposed by Blalock. Since the introduction of the aortic clamp, devised by Potts, Smith and Gibson, it is evident that this will add safety to division and suture of the ductus as the danger from hemorrhage from the aortic end should be almost eliminated.

The outstanding work of Maud Abbott in showing that 50 per cent of these patients die before the age of thirty, and outstanding reports by others, prove that this anomaly definitely shortens life. This is the prime reason for operation in an effort to prolong life expectancy.

Some of these cases develop bacterial endarteritis which is an indication for surgery, even should penicillin therapy be effective. The operation of Potts in anastomosing the aorta to the pulmonary artery produces a patent ductus syndrome and undoubtedly some will develop an endarteritis. So far none have been reported in the Blalock or Pott's operations. It will be interesting to see what this percentage will be, but even if it runs 5 to 10 per cent, that is no contraindication for relieving the cyanosis and improving the heart status in the Tetralogy of Fallot.

Surgery is usually best carried out between the ages of three and twelve, and in experienced hands gives excellent results with a low mortality.

Closure of Cavities in Pulmonary Tuberculosis Produced by Immobilization of Both Lungs*

JAMES H. CULLEN, M.D., F.C.C.P., ROBERT K. MYERS, M.D.,
ALVAN L. BARACH, M.D., F.C.C.P., and
GEORGE FOSTER HERBEN, M.D., F.C.C.P.
New York, New York

Introduction

Although striking medical advances have recently taken place in the treatment of infection by antibacterial agents, the use of rest in chronic pulmonary tuberculosis is still of great importance. In this paper the initial results of treatment with the lung immobilizing chamber will be described in 7 patients with persistent pulmonary cavities. In previous reports the principles by which complete lung rest was produced with simultaneous maintenance of normal pulmonary ventilation have been described.[1-8] Recent summaries[9,10] of the clinical results of treatment in 12 cases of advanced tuberculosis showed that 6 obtained arrest of their disease with ability to work, 1 was markedly improved; 3 showed slight to moderate temporary benefit and no change occurred in 2.

Local rest of the lungs was achieved in this method without collapse therapy or alteration of the normal lung or chest structure. The mechanism by which adequate pulmonary ventilation was provided included use of the principle of alternating pressure, described by Thunberg[11] as a method of resuscitation. By mechanically varying the pressure within an enclosed chamber approximately one-seventh of an atmosphere 24 times a minute, air was compressed and decompressed within an enclosed space in which the patient lay, resulting in a tidal air of approximately 300 cc. In order to prevent an earlier arrival of pressure to the outer chest wall than to the inner surface of the thorax the resistance of the respiratory passageway had to be counterbalanced in a precise manner; in the final arrangement the incoming air was led into the head chamber and then, momentarily delayed by a partition, entered the body compartment.[1-8] Cessation of lung movement was thus accomplished by simultaneously equalizing the pressure on both sides of the chest wall,

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(From the Department of Medicine, College of Physicians and Surgeons, Columbia University and the Presbyterian Hospital, New York, N. Y. and the House of Rest, Yonkers, N. Y.).

the chamber being described as an equalizing alternating pressure chamber, or, recently, as the lung immobilizing chamber.

The partition, which surrounds the neck of the patient, is lowered to produce differential pressures between 4 and 12 cm. of water. In most cases, the addition of 5 cm. pressure during the positive cycle in the head end of the chamber is adequate to overcome the resistance of the tracheo-bronchial tree. In patients with pulmonary fibrosis or those in whom narrowing of the bronchial tree has taken place as a result of chronic inflammation, higher differential pressures are required. When marked pulmonary emphysema is present, it is impossible to delay the air wave long enough to achieve a simultaneous application of equal pressures to both surfaces of the chest wall. At present this therapy is not attempted when a pressure higher than 15 cm. of water is required to overcome tracheo-bronchial resistance.

Method of Treatment

The method of treatment with the lung immobilizing chamber has become simplified so that it can be carried out at a private residence as well as at an institution. Of most importance is the training of the patient during the first week, during which he learns to stop breathing without movement of the chest. Subsequently, the chest is immobilized *without* conscious or voluntary effort for four months or longer. However, in cases in which inadequate supervision is given during the first week, spontaneous breathing may take place irregularly thereafter.

When the patient is placed in the chamber the pressure is turned to plus and minus 55 mm. of mercury at a rate of 26 times a minute. During the first period of ten minutes in the apparatus the patient is told to take three or four breaths, to exhale normally and then cease breathing. At the end of a minute or two he is apt to breathe, when he is told to take three or four breaths again and then stop breathing, with the suggestion that he will not require any voluntary breathing. During this time the chest is observed with the water manometer set at 5 cm. pressure. In most patients the first learning period is not prolonged beyond one hour. If it should be observed that the patient tends to enlarge his chest during the cycle of negative pressie, i.e., when the pressure indicator is turning from the positive to the negative side, he is asked to take short breaths during the opposite cycle of pressure, namely, the positive cycle. This is usually accomplished when the operator raises his hand as the pointer leaves from the negative to the positive side and says, "Breathe in." That method is usually not necessary except in cases that have a considerable amount of pulmonary fibrosis. During the follow-

ing week the amount of time in the chamber is increased one hour a day and at the end of ten days the patient is placed on a schedule of ten hours daily, usually three hours in the morning, 4 hours in the afternoon, and three hours in the evening.

During the first week costal respiration is dispensed with and in the following week the absence of diaphragmatic movement is noted. It is unnecessary to lecture the patient on persistent movement at the lower border of the ribs during the first week as this spontaneously disappears at the end of seven to ten days. There may be a slight movement observed over the surface of the abdomen as a result of compression of gas in the intestines; it is not a sign of movement of the diaphragm as this pressure is counteracted by an opposing pressure on the upper surface of the diaphragm from air entering the chest, as has been previously demonstrated by repeated x-ray exposures on the same film in these patients.

The effect of alternating pressure on the ear is similar to that experienced in a rising and descending elevator; it may at first be uncomfortable but in our patients it has not persisted as a source of discomfort after the first day or two. Congestion in the sinuses may temporarily take place but it also is not a handicap to the treatment. In a few instances the therapy has been discontinued for one or two days and recommenced without further difficulty. No other side-effects have been encountered except the unique development of mental and bodily relaxation that generally takes place in the chamber.

When patients have learned to dispense with voluntary breathing, the impulse for spontaneous muscular movement is remarkably diminished; in some cases no movement of the arms or legs is observed for hours at a time. It is of considerable interest that boredom is not complained of and that patients are for the most part willing to carry on the treatment without entertainment. In a few cases the radio is desired and ear-phones are used with them. When the patients observe that their chest is not moving, by placing their hands on the thorax and abdomen, their own faith in the chamber increases and their morale during the treatment is usually high. The feeling of claustrophobia may be anticipated by the patient, but as soon as ventilation of their lungs is performed by the apparatus without voluntary breathing this notion completely disappears. In no patient has it been a problem.

Although ten hours a day of residence in the chamber is considered the optimum time, recovery took place in one patient following a course of eight hours a day for three months. However, one patient, who had a large cavity in the left upper lobe, was treated for five hours a day, in an experimental trial of the

duration of treatment, without a favorable result. A succeeding trial of ten hours a day was followed by closure of cavity with the development of negative sputum in a period of four months. Since absence of lung expansion prevents inspiratory enlargement of the cavity, the check-valve mechanism responsible for maintenance of cavity in some cases is eliminated only during the time in the chamber. In projected investigation the value of longer periods of lung immobilization will be tried.

The patients are generally advised to take a high protein, low carbohydrate diet, as recommended by Sandler and Berke.[12]

Results

The case histories of the patients treated in this series will be presented. In 6 of 7 completed cases the treatment resulted in closure of cavity with negative sputum. In all these patients pneumothorax had been tried, either without initial success, or with reappearance of cavities following termination of pneumothorax.

CASE REPORTS

Case 1: Woman, age 27. The diagnosis of pulmonary tuberculosis was made when the patient was 17 years old at which time a right therapeutic pneumothorax was induced because of a large apical cavity. One year later the development of a fresh cavity on the left side was followed by the maintenance of bilateral pneumothorax. Three years before admission the left pneumothorax was abandoned and one and one-half years later a cavity appeared at this site. After a period of nine months she was admitted to the Presbyterian Hospital. Physical examination revealed evidences of involvement of each apex, but only the left side appeared active. Planigraphic x-ray films revealed a cavity 4 cm. in diameter at the left apex with a second cavity below and contiguous to it, 2.5 cm. in diameter, near the hilar region, in addition to many areas of fibro-calcified disease. Sputum was positive for tubercle bacilli, the sedimentation rate was 20 mm./hr. She was afebrile.

The pneumothorax on the right side was allowed to re-expand and chamber treatment was started. After four days of residence in the chamber the patient was completely immobilized. At the end of four months treatment, ten hours daily, the excavation on the left side was no longer visible by stereoscopic and planigraphic x-ray films. The concentrated sputum was negative for acid-fast bacilli and then all cough and expectoration disappeared. The patient was released to continue bed rest at home and she has remained symptom-free with no change in the x-ray examination of her chest during the following sixteen months.

Case 2: Woman, age 38. The patient had had diabetes since the age of 24. Twelve years ago pulmonary tuberculosis was diagnosed, for which a right therapeutic collapse was induced, and, shortly thereafter, collapse of the left side, because of cavitation in the left upper lobe. Two and one-half years ago a cavity appeared on the left side following termination of pneumothorax four years previously. A phrenic crush was performed which was followed by no change in the disease and one year

later she was admitted for chamber therapy. The sputum was positive for tubercle bacilli. X-ray examination of the chest revealed a cavity 3 x 4 cm. in the left posterior axilla which contained a fluid level. The patient ran a minimal fever of 99.6 to 100° F. by rectum. Sedimentation rate 25 mm./hr. Immobilization of both lungs took place promptly on the first day in the chamber. After two months the cavity had decreased to one-third its former size. Because of the persistence of a fluid level the patient was treated with inhalation of nebulized 1 per cent neo-synephrine, 0.5 cc. t.i.d. At the end of 18 weeks' treatment, 10 hours daily, the cavity had been replaced by a dense shadow approximately 1 x 1 cm. in diameter at the extreme periphery of the left upper lung

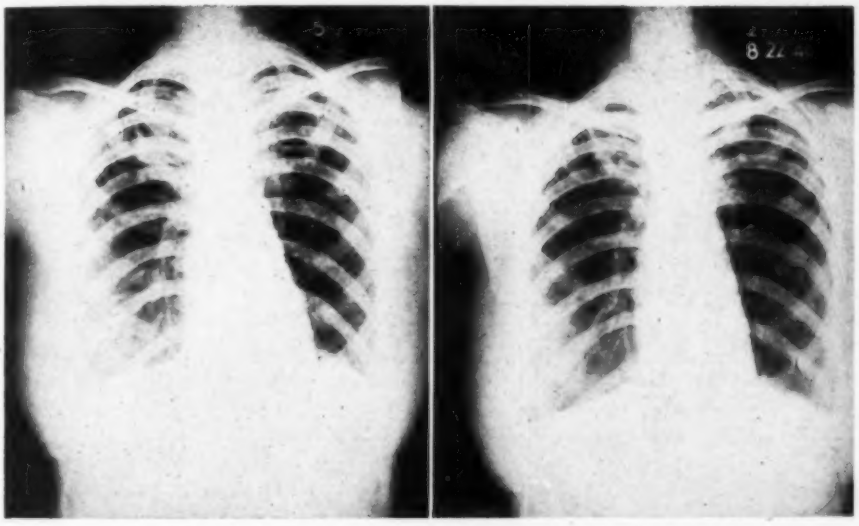

FIGURE 1: X-ray films before and after 3½ months treatment.

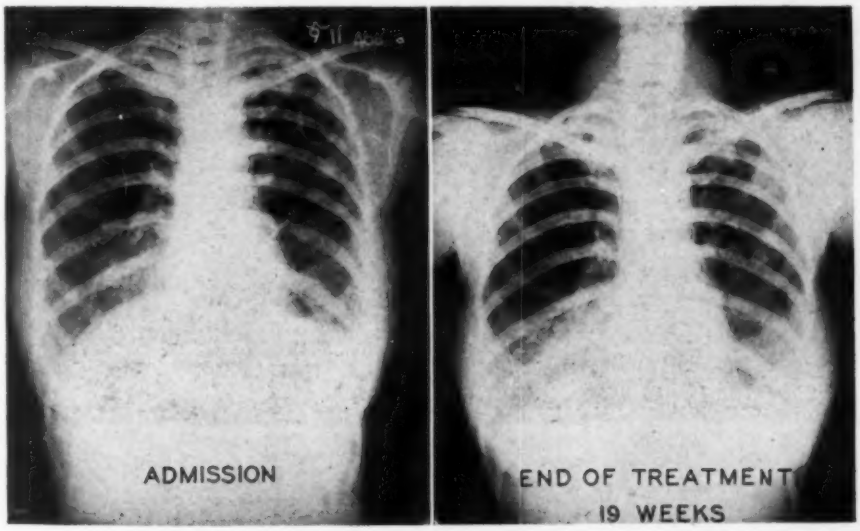

FIGURE 2: X-ray films before and after 19 weeks treatment.

field. Sputum and gastric lavages were negative for acid-fast bacilli. Films taken three, four and five months after discharge from the hospital showed no significant change but x-ray films 7 months after discharge showed a disappearance of the above described shadow. The patient has no cough or expectoration.

The disappearance of the two cavities in the first case, as revealed by the x-ray photographs (Fig. 1), is an illustration of the favorable result of immobilization of the lungs in a patient in whom previous bed rest had been followed by no improvement. In the second case, as the cavity became smaller, a density in the x-ray film which eventually replaced the rarefaction finally disappeared. The accompanying stereoscopic and planigraphic x-ray films (Figs. 2 and 3) illustrate the changes mentioned above.

Case 3: Man, age 30 years, was found to have tuberculosis one year and eight months before treatment. A cavity was noted in the right upper lobe with some extension to the left mid-lung. He was treated at first with rest and pneumothorax on the right, and later on the left, but adhesions prevented a satisfactory collapse and sputum remained positive. Ten months later he was admitted to the House of Rest where a 4 cm. cavity was found in the first costal interspace on the right and moderate infiltration in the left mid-lung field. The bilateral contraselective pneumothoraces were abandoned and the lungs re-expanded. Patient was afebrile.

The patient was quickly immobilized and at the end of a week was in the chamber ten hours a day. The cavity in the right lung gradually diminished in size, the sputum was decreased, but continued to be positive until he had five months of treatment when it became persistently negative. The cavity had disappeared, treatment discontinued; the patient was on bed rest for four months, ambulatory for eight months.

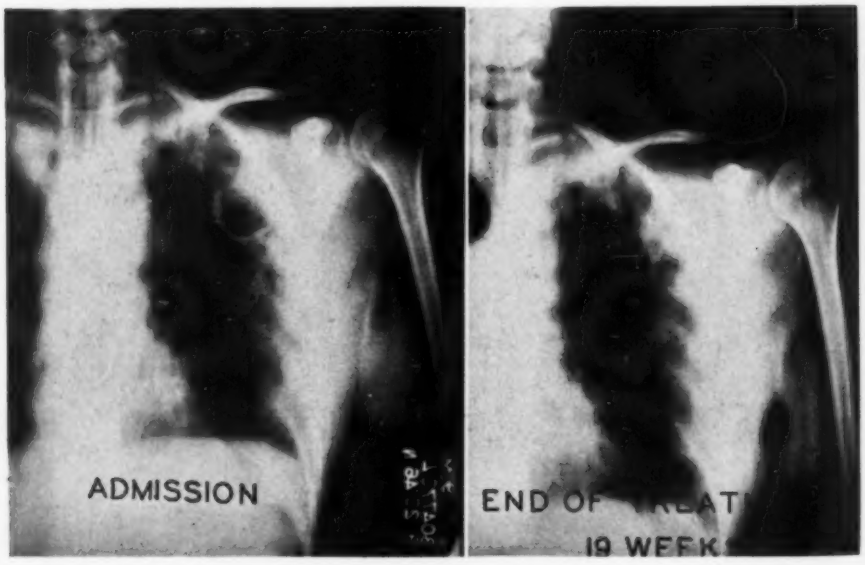

FIGURE 3: Planigraphic x-ray films before and after 19 weeks treatment.

Case 4: Woman, age 26 years, who had a history of tuberculosis for two years. Pneumothorax was attempted on the left without success, but the patient recovered under conservative therapy and was apparently well for one year and nine months when the disease in the left lung reactivated and revealed a cavity with positive sputum. She was admitted to the House of Rest two months later where a cavity 3 cm. in diameter was found in the second interspace on the left with positive sputum. The patient was afebrile. Since the cavity did not respond to bed rest she was begun on immobilizing lung treatment six months later. She became immobilized in one week. The sputum became negative two weeks after treatment was started and has continued to be negative. At the end of five months the cavity is no longer visible. A small shadow about 4 mm. in extent was seen in the x-ray at the site of the original cavity. She was on bed rest for four months, now ambulatory.

Case 5: Woman, age 26 years, whose pulmonary tuberculosis was diagnosed following a hemoptysis five years prior to admission. At that time she was found to have pulmonary tuberculosis with bilateral cavitation. Pneumothorax was induced on the left and six months later on the right. The bilateral cavitation fluctuated markedly in size and the patient's sputum continued positive for a year when it became negative for one year, after which time the cavity in the left lung reopened. The pneumothorax on the left, and a year later, because the cavity in the right reopened, the right pneumothorax were abandoned. At the time of treatment she had a 6 cm. cavity in the right mid-lung at the level of the third inter-costal space and a 3 cm. cavity in the second left costal interspace. The sputum was positive. Although considerable reduction in respiratory motion was obtained, complete immobilization was never secured because of pulmonary emphysema. She was, however, given chamber treatment. The cavity in the right lung finally reached one-quarter of its previous size, but the cavity in the left lung which was at first stationary began to increase. The chamber treatment was finally abandoned at the end of six months.

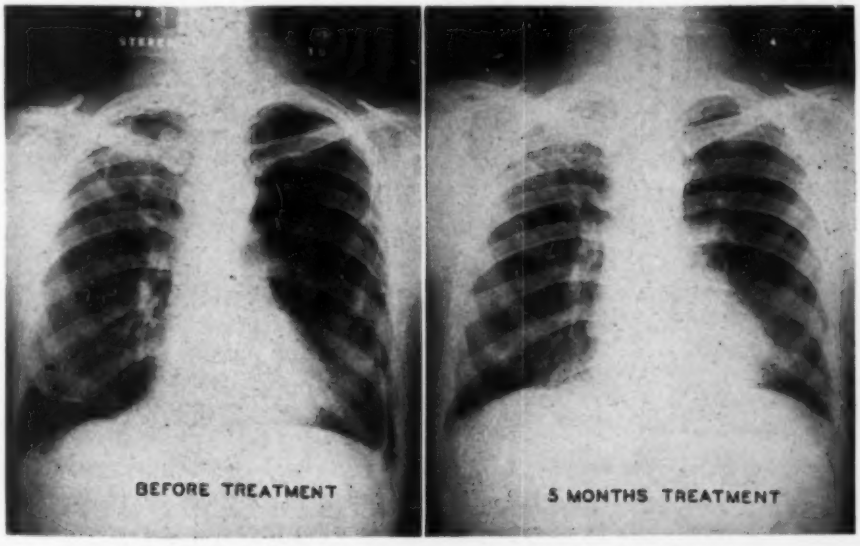

FIGURE 4: X-ray films before and after 5 months treatment.

The accompanying x-ray films (Figs. 4 and 5) illustrate the closure of cavity as a result of immobilizing lung treatment in Cases 3 and 4.

Case 6: Woman, age 52 years. The diagnosis of pulmonary tuberculosis was made six years ago. Pneumothorax was induced on the left at that time and was successfully maintained for two years when it was optionally abandoned. About three years later the disease in the left lung

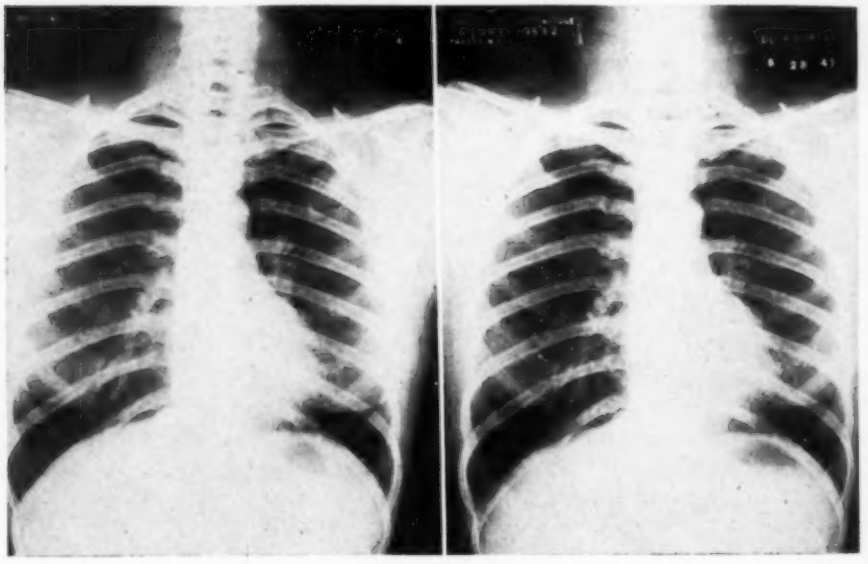

FIGURE 5: X-ray films before and after 4½ months treatment.

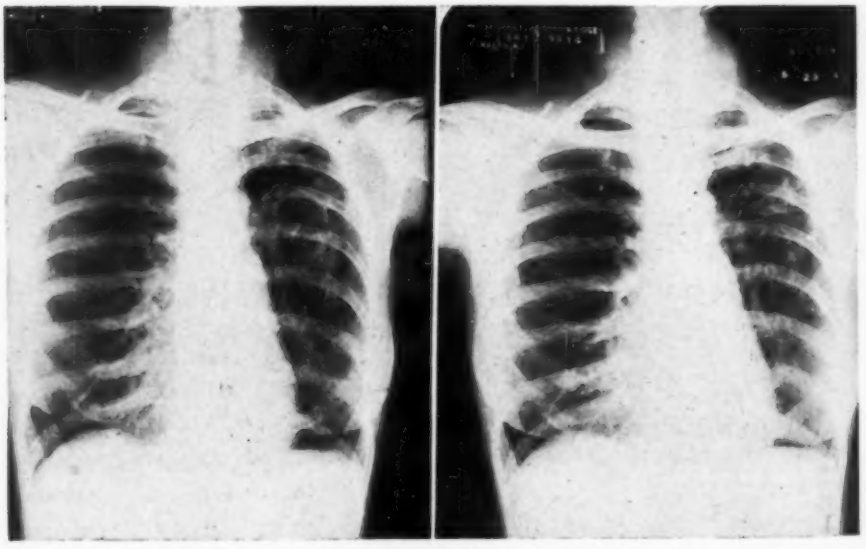

FIGURE 6: X-ray films before and after 4 months treatment and 2 months bed rest.

became re-activated with evidence of a cavity in the third interspace and the sputum was positive. She was admitted to the House of Rest where it was found that a small scattered nodulation extending from the first to the third intercostal space was present on the left with a 4 cm. cavity over the second rib. The patient was afebrile.

Pneumothorax was unsuccessfully attempted and after a failure to respond to bed rest the patient was treated in the immobilizing lung chamber for a period of four months at which time the cavity became no longer visible as seen in Fig. 6. The sputum was negative. She was on bed rest for four months, now ambulatory.

Case 7: This case was previously reported in part at the conclusion of two and one-half months treatment.* He has continued to have negative sputum tests for two and a half years.

*Barach, A. L.: "Continuous Immobilization of the Lungs by Residence in the Equalizing Pressure Chamber in the Treatment of Pulmonary Tuberculosis," *Dis. Chest,* 12:3, Nov.-Dec., 1946.

DISCUSSION

The patients who have been treated under our supervision since the last report include 8 cases of pulmonary tuberculosis with cavitation. The treatment was given an adequate trial in 7 in whom a complete course of 4 to 5 months was maintained. Of these 7 cases, 6 showed a disappearance of cavity with negative sputum during or at the end of treatment. Since bed rest and pneumothorax were not effective in accomplishing closure of cavity in these cases, the consistent response to total lung rest produced by the equalizing pressure chamber indicates that the method has unique value in the therapy of pulmonary tuberculosis. It may be pointed out that immobilization of the lungs in the equalizing pressure chamber entails none of the possible complications of collapse therapy. There are no harmful side-effects of its use. Furthermore, no loss of respiratory function takes place since there is no disturbance of the normal anatomical structure of the healthy as well as the diseased portions of the lungs. Most of the cases (6 of 7) in this series were those in which cavities were present without the extensive bilateral lesions present in the group of cases previously reported.

Although the exact mechanism by which deflation of the cavity takes place during residence in the lung immobilizing chamber cannot be definitely stated, it seems justifiable to point out that the absence of expansion of the chest is of crucial importance. In those cases in which a check-valve is responsible for a readier inlet of air into the cavity during inspiration than an outlet of air during expiration, the prevention of recurrent cavity enlargement appears to be a reasonable explanation for improvement. Other factors may be involved for which there is at present insufficient evidence to discuss adequately. For example, the degree

of ventilation of the cavity as well as the tension of oxygen and carbon dioxide in the cavity under conditions of this type of ventilation cannot be determined. In some cases, the lumen to a tuberculous cavity may be so small as to result in lessened ventilation during chamber therapy than takes place when normal inspiratory enlargement of the chest expands the cavity as well as the bronchial tube leading to it. In this series obstruction to drainage did not occur in such a way as to lead to the development of a fluid level although this took place temporarily in one patient described in a previous series.

The effect of lung ventilation without lung movement on body rest is conspicuous, since these patients lie more quietly than they could otherwise with ordinary bed rest. The suggestion that patients might lie as quietly in a chamber of this sort without being exposed to the mechanism of equalizing pressure therapy has been shown to be impossible. Patients who lie in the chamber without lung rest mechanism being activated show the same movement from side to side as they do at bed rest. The complete form of body and lung rest obtained in the lung immobilizing chamber has an added value in that the total oxygen consumption is (presumably) markedly decreased. It may be emphasized that this form of body rest is not possible in the presence of voluntary respiration, since prolonged observation of patients who attempt rigid body rest reveals unmistakably that the degree of movement of the voluntary musculature is far in excess of patients in the equalizing pressure chamber during treatment.

The special character of the pulmonary, bodily and mental rest that takes place during residence in the chamber manifested by the lowering of the temperature, between 0.5 to 1° F., that is generally manifested even in patients without fever. In addition, the drop in pulse rate from 7 to 20 beats per minute suggests a lowering of the total oxygen consumption. In one patient who was very carefully observed the temperature range from 8 a.m. to 8 p.m. before treatment was from 97.6 to 99° F. by mouth and at the end of three weeks ranged from 96.4 to 97.8° F. The blood pressure at the end of one hour of residence in the chamber is generally lowered from 15 to 20 mm. Hg. in the systolic phase and from 5 to 10 mm. in the diastolic phase.

The electrocardiogram also shows a uniform elevation of the T wave in leads 1, 2 and 4; this is present both in the cases that have a marked as well as those that have a small decrease in pulse rate. The increase in height of the T wave, between 0.5 and 2 mm. persists throughout the period of residence in the chamber and disappears gradually during a period of one-half hour after removal from the chamber. The average elevation of

T1 and T2 in 40 tests on 5 patients was 0.8 mm. The mechanism of elevation of the T wave will be commented upon when these data are presented in full. Whether an increase in oxygen tension of cardiac muscle takes place as a consequence of a maintained normal arterial oxygen saturation and a diminution in oxygen consumption of cardiac tissue is a matter of speculation. It would seem likely that the total cardiac output was decreased, along with the total oxygen consumption.

The absence of recurrent inspiratory negative chest pressures, ordinarily useful in aspirating blood into the right heart, does not appear to have an embarrassing effect on the circulation; perhaps the total blood flow is maintained at a higher level of intra-auricular pressure. In any event, this form of lung and body rest, accompanied by mental relaxation, is accompanied by alterations of physiologic equilibria, the precise study of which may reveal findings of considerable interest.

SUMMARY

The mechanism by which rest of both lungs is obtained without collapse therapy in subjects with pulmonary tuberculosis is described.

The clinical results of seven patients with cavitary pulmonary tuberculosis treated with immobilizing lung chamber therapy are reported.

In 6 of 7 completed cases, disappearance of cavity by x-ray examination and the development of negative sputum took place in a period of 4 to 5 months' treatment.

Immobilization of both lungs provides a therapeutic method of treatment of pulmonary tuberculous cavities that is free from the risk of the harmful side-effects of collapse therapy. The *absence* of respiratory enlargement of the lungs, which expand the tuberculous cavity in normal respiration, may be in part the mechanism of the gradual deflation of pulmonary cavities in patients treated in the immobilizing lung chamber. This mechanism would be particularly applicable in cavities with a small bronchial passageway in which a check-valve mechanism was previously operative.

This method of treatment requires careful supervision during the first week or ten days of the learning period. Thereafter, the properly trained patient generally maintains continuous arrest of lung movement without conscious effort.

RESUMEN

Se describe el mecanismo mediante el cual se obtiene el descanso de ambos pulmones en tuberculosos pulmonares sin emplear la colapsoterapia.

Se describen los resultados clínicos obtenidos en siete tuberculosos pulmonares con cavernas, tratados en cámaras de inmovilización del pulmón.

En 6 de los 7 casos terminados desapareció la caverna en la radiografía y se volvió negativo el esputo en un período de tratamiento de 4 a 5 meses.

La inmovilización de ambos pulmones proporciona un método para el tratamiento de cavernas pulmonares tuberculosas exento del riesgo de los efectos secundarios perjudiciales de la colapsoterapia. La *ausencia* de expansión respiratoria de los pulmones, que agranda la caverna tuberculosa durante la respiración normal, puede ser parte del mecanismo por el cual se desinflan gradualmente las cavernas pulmonares en pacientes tratados en la cámara de inmovilización del pulmón. Este mecanismo sería aplicable particularmente a cavernas con un pasadizo bronquial pequeño, en las que previamente existía un mecanismo de válvula de retención.

Este tratamiento requiere cuidadosa vigilancia durante los primeros siete o diez días del período de aprendizaje. Después de eso el paciente propiamente adiestrado generalmente mantiene la continua suspensión del movimiento pulmonar sin esfuerzo consciente.

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Discussion

A. A. LEONIDOFF, M.D., F.C.C.P.
Poughkeepsie, New York

Physiologically the principle of Dr. Barach's treatment is correct. He devised a lung equalizing machine which "breathes" for the patient, who remains still without expansion or re-expansion of the lungs. At the Nettie Bowne Hospital in Poughkeepsie, New York, we have two chambers and I would like to present the case studies of two patients. While in the chamber the patient is perfectly contented, happy and relaxed and takes the long, and what appears to be arduous task of lying still for a stretch of ten hours cheerfully.

Mrs. A. T., 32 years old, was admitted to our sanatorium in January 1945 with the diagnosis of far advanced pulmonary tuberculosis, involving right upper lobe with giant cavity and slight scattered infiltration throughout the left upper lobe. She had cough, positive sputum and occasional elevation of temperature. On absolute bed rest she gained in weight and strength, her temperature became normal, other symptoms lessened and she was allowed limited privileges up and about her room. However, in the summer of 1945 there was spreading of disease in both lungs. On absolute bed rest her clinical picture again improved. Surgery for the right upper lobe was considered but she refused it. Improvement continued until March 1946 when the disease again spread, especially in the right upper lobe. Her temperature increased and she developed tuberculous laryngitis in May 1946. She continued to lose ground. Her sedimentation rate was high and cough and expectoration increased with continued positive sputum. She was placed in the chamber in August 1946 and in four weeks the symptoms markedly diminished. After a total of 20 weeks of continuous chamber treatment, the cavity in the right upper lobe remained unchanged, and there was no evidence of activity in the left lung. The previously recommended surgery was performed and at the present time she is in good condition. This case shows the value of the treatment especially in regard to clearing the disease in the left lung so that she was able to undergo thoracoplasty on the right side.

Mrs. M. T., 44, gave the history of having had tuberculosis diagnosed in September 1945, but according to her statement she unquestionably had the disease long before that time. When she was admitted to our institution in January 1946, she had far advanced bilateral pulmonary tuberculosis with a cavity (3" x 2" in diameter) in the 1st and 2nd interspaces on the left side. Absolute bed rest was instituted but no definite improvement occurred until November 1946 when some symptoms diminished, but the sputum was still positive. The cavity was somewhat smaller but there was increase of the process in the left central lung field, and the lesion in the right upper lobe was more pronounced. Later the cavity appeared somewhat smaller than on previous observation. The right upper lobe showed some honey-combing. Her temperature again became elevated to 101 with a corresponding pulse of 104. It was

then decided that she should be given chamber treatment. After five weeks of this treatment only a small remainder of cavity in left upper lung field was seen, but there was still infiltration throughout the left upper lung field. The right lung showed decrease of involvement. Her temperature became normal, cough was practically gone and expectoration ceased. The sputum was negative, but the sedimentation rate was still 32 mm. (Cutler). This patient stayed in the chamber until June 1, having a total of 985 hours of treatment. At this time she had no symptom or complaint. The sedimentation rate was normal and her sputum was negative. Gastric lavage was done and reported negative on direct smear and culture. Guinea pig innoculation was done but to-date no report has been received. In the left lung the cavity could not be visualized. Some residual infiltration was still present and perhaps some honey combing. In the right lung the lesion in the 1st and 2nd interspace was practically gone and in the 3rd interspace markedly decreased. It was decided that to achieve further recovery, the patient should have another ten weeks of treatment, but she failed to comply. This case represents remarkable improvement of general physical condition as well as x-ray changes in twenty weeks and in comparison with the previous 16 months of bed rest, the gain was evident.

Only the future will tell the final results. We strongly believe in the validity of this treatment and wonder what would happen to cavities of short duration with this type of treatment. The two cases here cited had far advanced, hopeless disease.

It is a pleasure and an honor to discuss the paper of such a distinguished scientist as Dr. Barach and before a gathering such as we have here today.

Discussion

MINAS JOANNIDES, M.D., F.C.C.P.
Chicago, Illinois

In pulmonary tuberculosis rest is the fundamental basis of all treatment. "Physical and mental rest" are the routine instructions of all phthisiologists to their patients. Collapse therapy measures are primarily devised to promote additional local rest to the lung. Recently Dr. Barach perfected his differential pressure lung immobilizer which we have been using for about eleven months, at the Alexian Brothers Hospital, Chicago, Illinois.

It is indeed a privilege to discuss Dr. Barach's paper on his more recent results with the use of his chamber. Dr. Barach has supplied for the tuberculous patient an additional step in our efforts for his recovery. It is obvious that with the large amount of elastic tissue in the lung, it will have a tendency to contract when it is immobilized. It is for this reason that cavities close

also with the use of practically all forms of collapse therapy. In our use of the chamber we have adopted the principle of giving assistance to patients in whom the accepted methods of therapy are either contraindicated or have been of no value. We have used the lung immobilizer as a preoperative and postoperative measure. Preoperatively, pulmonary rest by immobilization enhances the chances for recovery and reduces morbidity to a minimum. Postoperatively, especially after thoracoplasty, the chest wall is protected against paradoxical mobility and a better result may be obtained.

We have used the immobilizer on thirteen patients, two of whom had to be discontinued; one because of claustrophobia and the other because of severe pain in the ears due to occluded eustachian tubes. In the remaining eleven patients we invariably noticed improvement, with conversion of the sputum in nine patients. Dr. Barach mentioned the tendency of the patient to relax, both in the chamber and for an hour or two after leaving the chamber. We have noticed the same reaction in our patients and often we were requested by them for longer periods in the chamber than eight hours.

The possibilities for the use of the immobilizer are many and varied. At present only tuberculous cases are being treated. I anticipate its use in arterial hypertension, cardiac disease and perhaps even possibly in cases of toxic goitres.

As more chambers are produced, this excellent method of treatment will become available to more and more persons who at present are doomed and who may have a chance to prolong their lives and become rehabilitated to a more useful and happy existence.

Pulmonary Heart Disease*

W. A. SODEMAN, M.D.**
New Orleans, Louisiana

Although pulmonary heart disease, or cor pulmonale, is generally conceded to result from pulmonary arterial hypertension, the latter more frequently produces cardiac damage when the term cor pulmonale is not used. Figure 1 gives a classification of the conditions producing such hypertension. Most common are the causes falling under Ib, Figure 1, in which obstruction is transmitted backward from structures beyond the pulmonary tissues. Thus, all conditions which affect and cause failure of the left ventricle, such as systemic arterial hypertension and coronary sclerosis as well as obstructive lesions in the left heart, mitral stenosis, for example, are etiologic factors. Shunts, II in Figure 1, connecting the pulmonary artery and aorta, act in a similar way when they are of sufficient magnitude physiologically to produce significant change in pulmonary pressure. Patent ductus arteriosus is an example of such a chronic condition, and aortic aneurysm rupturing into the pulmonary artery an example of an acutely developing one.

Cor pulmonale, as the term is usually used, is concerned only with the recognition of those conditions falling under Ia in Figure 1, in which changes in the pulmonary structures themselves are responsible for the pulmonary hypertension and, in turn, for damage to the right heart. Of these only those which are chronic in type will be considered in this report. Acute damage to the pulmonary circulation of sufficient magnitude to produce acute right sided cardiac strain, as occurs in pulmonary embolism, will not be discussed.

Chronic cor pulmonale may be produced by a variety of changes in the structures of the chest. These have been classified by Spain and Handler[1] into three large groups, anatomic alterations of the thoracic cage, of the pulmonary vascular system, and of the pulmonary parenchyma. Those arising as a result of change in the lung constitute the largest and most important group.

In the past five years in the records of the Charity Hospital there have been 15 recorded instances of chronic cor pulmonale.

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**Department of Tropical Medicine and Public Health, School of Medicine, The Tulane University of Louisiana and The Charity Hospital of Louisiana at New Orleans.

The etiologic factors underlying this group were related to the presence of emphysema. In 8 instances bronchial asthma was present, and in 3 emphysema not related to a gross pulmonary diagnosis. In 2 tuberculosis, in 1 bronchiectasis, and in 1 schistosomiasis were present. If these are added to the larger groups of Scott and Garvin[2] and Spain and Handler[1] and the etiologic factors for the entire group listed, they will appear as shown in Figure 2. Perusal of this list will indicate the types of pulmonary disease usually responsible for chronic cor pulmonale. All of the conditions reported as causing chronic cor pulmonale are not included for some of the rarer ones, such as compression of the pulmonary artery by aortic aneurysm[3] and multiple cysts of the lung, did not occur in the group.

The mechanism whereby these conditions produce changes in the right heart will not be detailed here. Others[1] have recently discussed these aspects of the problem and the reader is referred to that report. Diffuse obstructive emphysema, either primary or secondary to such diseases as pulmonary tuberculosis or silicosis, is considered as the significant underlying pulmonary factor in the vast majority of the cases. This condition apparently produces a changed pressure relation within the alveolus and in this manner there results an increased resistance to the flow of

FIGURE 1

PULMONARY HYPERTENSION

I. Obstruction to pulmonary blood flow.
   a. *In the pulmonary structures themselves.*
   b. Transmitted backward from structures beyond the pulmonary tissues.

II. Augmentation of pulmonary pressure through shunts.

FIGURE 2

ETIOLOGIC FACTORS IN 123 COLLECTED CASES OF CHRONIC COR PULMONALE

Emphysema	75
Bronchial Asthma	14
Tuberculosis	9
Silicosis	8
Bronchiectasis	7
Silicotuberculosis	5
Pulmonary fibrosis	1
Kyphoscoliosis	1
Pulmonary arteriosclerosis	1
Organized pulmonary thrombi	1
Schistosomiasis	1

blood to the lungs. Such associated factors as anatomic obliteration of the pulmonary vascular bed, fibrosis of the lung, compensatory polycythemia and overfilling of the heart are considered as secondary aggravating factors of varying significance.[1]

A review of the clinical manifestations of chronic cor pulmonale in our group disclosed that 13 patients presented themselves with the picture of right ventricular failure. In 2, or 13.3 per cent, of the patients, the outstanding clinical manifestations were those of the primary pulmonary disease. Brill[4] in 1939 recognized the grouping of patients into these two classes, with what he called the early pulmonary phase and a later cardiac phase. Until right sided heart failure with distended neck veins, hepatomegaly, prolonged circulation time and dependent edema appear, the symptoms have been essentially those of the pulmonary disease, often with dyspnea, cough, and hemoptysis, findings which do occur with affections of the left side of the heart and which have been present in many of these patients for years. A change in the symptoms in these patients is an important evidence of oncoming cardiac failure. The dyspnea may be worse; the patient can no longer lie down; cyanosis, if not present, appears, and, if present, is accentuated. When the patient was examined, the pulmonary state, including emphysema, was usually present and the evaluation of the heart was difficult. This difficulty rested chiefly in the pulmonary changes which interfere with percussion of the cardiac borders. Heart tones, too, were frequently distant and various systolic murmurs, difficult to interpret clinically, were present. However, a number of findings were often present which frequently were disregarded. These will be mentioned below. When the signs of right sided heart failure, distended veins, hepatomegaly, and edema, occurred, these were, of course, evident.

It is clear that until frank evidences of heart failure appear, the possibility of a clinical cardiac state being present is frequently not entertained. This was true in our group and in one large series of 60 patients,[1] in not one case was the diagnosis of cor pulmonale established or entertained prior to the onset of the symptoms and signs of heart failure. The frequency of cor pulmonale in the necropsied series of Scott and Garvin[2] was 50 cases in 6,548 necropsies, including 890 involved cases of cardiac disease. This represents 6.3 per cent of all cases of cardiac disease necropsied. In our hospital, the clinical recognition of cor pulmonale represents less than one per cent of the cases of heart disease seen, indicating that in all probability many of the instances of cor pulmonale entering the institution are not diagnosed. Usually, when a diagnosis is made, in 86.7 per cent of our patients, the patient is already in heart failure, and treatment, which is

most important in the stage before heart failure develops, has been too long delayed.

When heart failure does appear, especially because of the age distribution of the patients, which in our group was greatest from 40 to 49 years, arteriosclerotic heart disease is a frequent diagnosis. This was true in our series and in that of Spain and Handler,[1] chiefly because in heart failure, in this age group, when the common causes for heart disease other than arteriosclerosis are eliminated, that diagnosis is often given on an exclusion basis. A review of the records from our patients in whom the diagnosis was finally made indicates that the delay in recognition of cor pulmonale is due, in large part, to neglect of a number of diagnostic aids. For example, the evaluation is delayed of many multiple small findings on history and physical examination, any one of which alone is difficult to interpret, but which, taken as a group, make a suggestive and, at times, a formidable story. Then, with such a story, the special procedures of electrocardiography, fluoroscopy, and the determination of venous pressure and circulation time can be utilized. Even with these aids, it may take, as several of our records clearly show, observation for months or several years to indicate definitely the presence of cardiac damage. To increase the frequency of such diagnoses, it is proposed that all patients with chronic pulmonary disease, especially when associated with emphysema, be examined carefully by these methods.

In physical examination, special thought must be given to the evidences of right ventricular strain. This includes, aside from the obvious evidences of edema, hepatomegaly, and distended neck veins, particular attention to early findings, accentuation of P_2, the pulmonic diastolic impact, observation of the precordium for a retraction wave, which often occurs normally over the right ventricle, but becomes more marked in right ventricular hypertrophy. It appears as a wave progressing across the precordium toward the apex and is often accompanied by a distinct systolic retraction in the epigastrium. This latter finding also occurs with a ptotic heart, in pleural effusion, and in anemias. All of these physical findings, each by itself of no great significance and frequently capable of being interpreted and explained on other grounds, when occurring together or in multiples, are of extreme importance for they then make the possibility of right ventricular disease as their cause more likely and should lead to further investigation of the patient by the methods about to be discussed. In 10 of our 15 patients, the diagnosis was first suggested on these grounds which led to electrocardiographic, radiologic, and hemodynamic studies.

Electrocardiographic studies, including chest leads, were done

in 12 of our 15 patients. In 2 instances the findings gave the first suggestions of the presence of cor pulmonale. This figure would have been higher had it not been for the alertness of the staff in establishing the physical evidences of right ventricular strain as described above. In 6 instances definite right axis deviation occurred; in 2 it was slight. In 3 patients, low complexes were present and in one nothing unusual at all. The presence and degree of right axis deviation, the determination of the axis index, the study of a series of chest leads taken from right to left across the precordium and the use of special chest leads are the most important points for consideration. The method of examination, particularly when serial tracings are available, may be of great help. This was true in 2 instances in our group.

Radiologic examination is practicable both with fluoroscopic and roentgenographic techniques. These methods, especially when the oblique views are used along with the PA views, will give much information concerning the right ventricle. In the PA view prominence of the region of the pulmonary conus may be seen at times when all other evidences are negative. In general, in our group, in which PA roentgenograms were taken, in 11 of the 15 patients prominence of the pulmonary conus was not sufficiently remarkable to give great help. However, when seen, even though only suggestive, it should lead to adequate oblique views or the use of fluoroscopy. In both oblique views encroachment of the heart shadow beyond the limits of normal may, at times, be seen long before other evidences of cardiac involvement occur. Fluoroscopy lends itself well to study of the oblique positions and we have found it most helpful in establishing the presence of right ventricular involvement. In 2 of our patients it was the only evidence present.

Hemodynamic studies by the venous pressure and circulation time determinations are simple clinical procedures. In the presence of emphysema the former may be difficult to interpret. The circulation time, especially in an alert patient, is much more frequently helpful. Of course, early in these pictures, at a time when diagnosis is most important, both these findings are normal. We have found the circulation time more reliable than the venous pressure studies in emphysema. In 2 cases, for example, with venous pressures of 110 and 130 mm. of water, values not absolutely abnormal, the circulation times, arm-to-tongue, were 30 seconds and 23 seconds, respectively.

RESUMEN

El análisis de los protocolos de 15 pacientes con cor pulmonale crónico indica que (1) la presencia de enfermedad pulmonar

crónica es una indicación para que se lleve a cabo una investigación completa del corazón del paciente a fin de verificar la posible presencia de cor pulmonale; (2) las manifestaciones clínicas sobresalientes desde el punto de vista de síntomas y signos físicos antes de que aparezca la insuficiencia cardíaca franca, son índices poco valisos de la presencia de esta enfermedad, pero signos tempranos en la historia y en el exámen físico de daño al ventrículo derecho, a menudo descuidados debido a su carácter menor, son indicaciones especialmente útiles en la investigación adicional del paciente; (3) en estas circunstancias es necesario llevar a cabo una investigación especial del corazón con estudios adecuados electrocardiográficos, roentgenoscópicos y hemodinámicos a fin de establecer el diagnóstico de cor pulmonale crónico con alguna exactitud en la etapa anterior a la aparición de insuficiencia del corazón derecho, la etapa más importante en el tratamiento.

SUMMARY

Analysis of the records of 15 patients with chronic cor pulmonale indicates that (1) the presence of chronic pulmonary disease is an indication to carry out a complete investigation of the patient's heart to establish the possible presence of cor pulmonale; (2) the outstanding clinical manifestations from the standpoint of symptoms and physical findings before frank heart failure appears are a poor index of the presence of this disease, but early historical and physical evidences of right ventricular strain, often neglected because of their minor character, are particularly helpful indicators for further investigation of the patient; (3) the addition of special investigation of the heart under the above circumstances with adequate electrocardiographic, fluoroscopic, and hemodynamic studies is necessary to establish the diagnosis of chronic cor pulmonale with any degree of accuracy in the stage before right sided heart failure appears, the stage most important in treatment.

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The Role of Bronchoscopy in Clinical Medicine and Surgery*

ARTHUR Q. PENTA, M.D., F.C.C.P.**
Schenectady, New York

Due to the demands of the rapid progress of medical science during the past decade, bronchoscopy has come to play an important role in the diagnosis and treatment of pulmonary diseases. To quote that famous teacher of bronchoscopy, Chevalier Jackson: " 'Look and see is the order of the day. In every accessible region of the body, the aid of direct vision is called upon to contribute to diagnosis and treatment."[1]

In the diagnosis of thoracic diseases, the internist, by means of external inspection, percussion, and auscultation, usually obtains sufficient information to establish a tentative diagnosis. His findings may be further corroborated by the roentgenologist, why by means of fluoroscopic and roentgenographic examinations has an opportunity, as it were, to look through the chest. But it remains for the bronchoscopist, by means of direct visual inspection, to look inside the bronchial tree and observe the underlying pathologic condition. It becomes evident, then, that when there exists such a close association between the internist, the roentgenologist, and the bronchoscopist, the diagnosis of pulmonary conditions is greatly facilitated.

Aside from the removal of aspirated foreign bodies in lungs, for which no procedure other than bronchoscopy is worthy of a moment's consideration, the profession at large has not realized that the greatest value of the bronchoscope is in diagnosis. A recent statistical survey of the patients admitted to several of the large bronchoscopic clinics in this country revealed that only 2 per cent of the admissions were for the removal of foreign bodies. The late Dr. Howard Lilienthal, one of the great teachers of thoracic disease, has on many occasions stated: "The scope of bronchoscopy has rapidly widened from the mere extraction of foreign bodies to the diagnosis and treatment of many pulmonary lesions, such as abscess of the lung, tumor of the lung, bronchial narrowing, bronchial ulceration, postoperative atelec-

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**From the department of Broncho-Esophagology, Ellis Hospital, Schenectady, New York.

tasis, and as an aid to the thoracic surgeon in the localization of thoracic diseases."

In the field of diagnosis the bronchoscopist by means of direct vision is able to inspect the inside of the bronchial tree and remove tissue for histologic examination, thereby establishing, in many instances, the early diagnosis of bronchial carcinoma. In a small number of cases in which the endobronchial tumor mass is located in the smaller bronchi, beyond the range of bronchoscopic vision, the bronchoscopist, by means of bronchial lavage of the involved bronchus followed by cytological examination of the aspirated solution (normal saline) may serve as a further aid in establishing the early diagnosis of bronchogenic carcinoma. By means of special aspirators introduced through the bronchoscope, it is possible to remove purulent secretions for the relief of bronchial obstruction. Also secretions removed by this method, being free of oral contamination, assure a more desirable bacteriologic study, and are of greater value if a vaccine is to be prepared.

That bronchoscopic examination is not a hazardous procedure is best illustrated by the fact that during the past ten years, during which period the author has personally carried out over 2,500 bronchoscopic procedures, the majority of the patients examined or treated were handled as out-patients.

Bronchoscopy and Diagnosis

Carcinoma of the Lung: A relatively common disease, carcinoma of the lung is responsible for approximately 10 per cent of all cancer deaths, an occurrence which is frequent enough to arouse interest on the part of the medical profession. Bronchoscopic examination with removal of the tissue for histologic study is by far the most important diagnostic procedure available, and if an early diagnosis is to be made, bronchoscopy should be employed early. In a recent study of the subject, Overholt and Rumel[2] call attention to the fact that approximately three-fourths of all primary lung tumors are situated in the major bronchi so that they are within range of bronchoscopic vision. His findings are in complete agreement with those of Jackson and Konzelmann,[3] who in a recent article stated that bronchoscopic biopsy was positive in about 75 per cent of the cases of bronchial carcinoma. In a series of 60 cases personally examined by the author during the past ten years, a positive biopsy was obtained in 42 cases. This high percentage of positive biopsies is definite evidence that bronchoscopic examination plays an important role in the diagnosis of lung carcinoma.

The symptoms produced in primary carcinoma of the lung depends to a great extent on the degree of bronchial obstruction.

By far the most important early symptom is a dry, hacking cough accompanied by slight bronchial wheezing. Physical examination during this early stage may reveal a few coarse moist rales and asthmatic wheezing over the involved pulmonary area. Roentgenographic studies of the chest during this stage may be entirely negative since the tumor is not large enough to cast a shadow. At this time the patient usually seeks medical advice because of the cough and wheezing. In spite of the fact that no improvement is usually shown after several weeks or several months of medical treatment, very little thought is given to the possibility of bronchial carcinoma.

Slight asthmatic wheezing is a very important early symptom. It is commonly present in all cases of bronchial carcinoma. Wheezing means only one thing—partial bronchial obstruction, which may be produced by a variety or combination of conditions, among which is bronchial carcinoma. The importance of this one symptom alone cannot be too strongly emphasized. In the series of 60 cases of bronchial carcinoma bronchoscopically examined by the author during the past ten years, it was astonishing to learn that 18 of the patients, because of recent wheezing and slight dyspnea, had been previously treated for bronchial asthma. Any patient, particularly of the cancer age group, presenting this symptom, should immediately by x-rayed and should have the benefit of a diagnostic bronchoscopy. The appearance of blood-streaked sputum or frank hemoptysis are also frequent early symptoms and are usually due to the trauma of coughing and ulceration of the tumor mass. Pain, as in other malignancies throughout other parts of the body, is definitely not an early symptom.

For the purpose of clinical study, Overholt divides the clinical course of primary lung carcinoma into the following divisions: (1) the stage before bronchial occlusion, (2) the stage of bronchial occlusion, and (3) bronchial occlusion with secondary infection.

It is important that in order to increase the operability of this condition, which in the past has had a mortality of 100 per cent, and in order to take advantage of the rapid progress already made to date in the field of thoracic surgery, the diagnosis should be established early, before the stage of bronchial occlusion. By far the most important diagnostic procedure available at the present time is bronchoscopic examination (Figs. 1, 2 and 3).

Benign Neoplasms of the Tracheobronchial Tree: Without the aid of bronchoscopic inspection, the diagnosis of benign neoplasms of the trachea or bronchi would probably never be made except by inference. The symptoms and physical signs produced depend to a great extent on the degree of bronchial obstruction

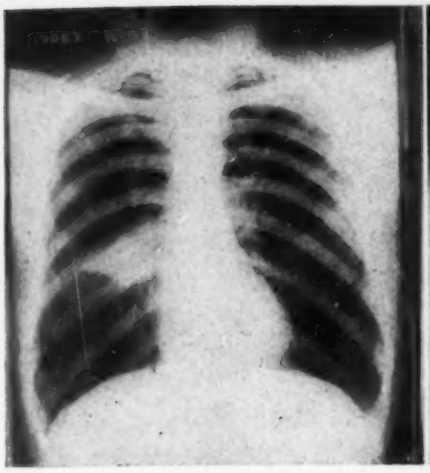


FIGURE 1

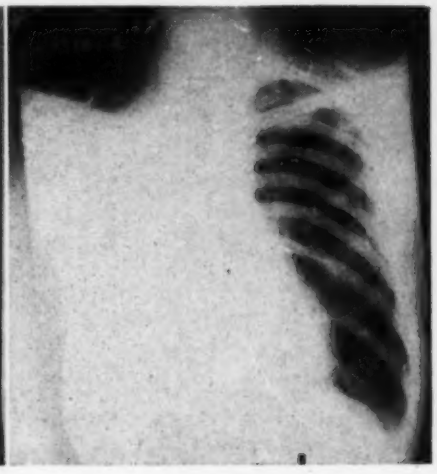


FIGURE 2

Figure 1: Radiograph of chest of male patient age 42, showing a lesion in the mid-region of the right lung. For the past three months the patient's chief complaint was coughing and bronchial wheezing. Bronchoscopic examination revealed a small tumor mass of the right main stem bronchus which encroached upon the orifice of the middle lobe bronchus. Biopsy taken of this tumor mass was reported as an epidermoid carcinoma, Grade 2. He was advised to undergo a thoracic operation and in October 1942, Dr. Ralph Adams performed a total pneumonectomy of the right lung.

Figure 2: Radiograph taken in 1947, showing the characteristic appearance of chest following total pneumonectomy. For the past two years the patient has been steadily employed and except for slight dyspnea upon exertion, he is otherwise in good health. Periodic x-ray studies during the past five years revealed no metastasis.

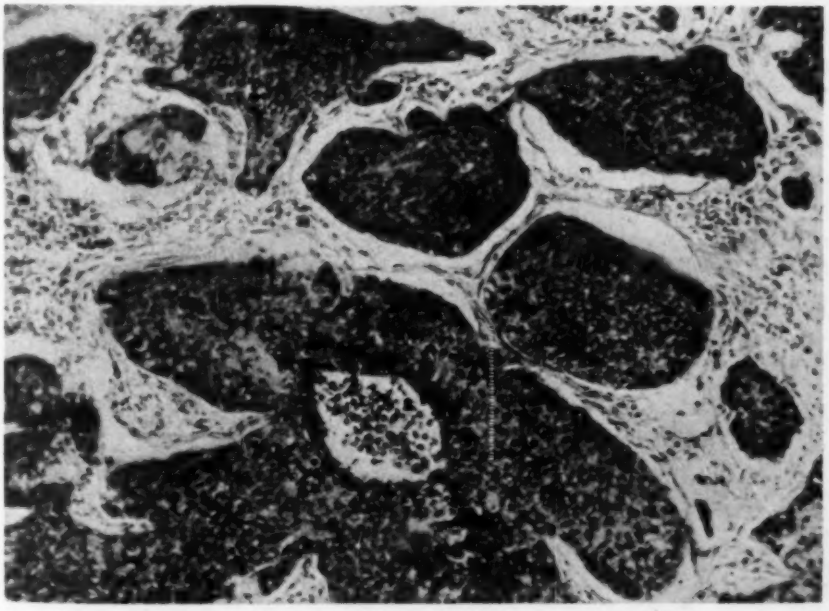


FIGURE 3: Histological section from removed lung showing epidermoid carcinoma.

and the presence of infection. It is always to be remembered that the first symptoms produced in early bronchial obstruction, regardless of the cause, are those of wheezing and coughing. If coughing is associated with blood-streaked sputum, bronchial neoplasm should always be considered in the differential diagnosis. Benign tumors of the trachea or bronchi can usually be successfully removed by bronchoscopic procedure. It is important that the diagnosis be established early, before any degree of bronchial obstruction and pulmonary infection has taken place.

Tumors of the trachea, particularly those pedunculated in type, may obstruct the egress of air, resulting in severe bilateral emphysema. As the tumor mass grows in size it may completely block the trachea causing sudden asphyxia. Because of the asthmatoid-like wheezing and emphysema which is usually present, many of these patients give a history of having been treated for bronchial asthma.

Bronchial Stenosis: This condition results from (1) inflammatory reaction produced by foreign bodies, (2) specific infections such as syphilis and tuberculosis, or (3) con genital tendencies. The clinical symptoms and physical signs produced are chiefly those of bronchial obstruction. Once the preliminary physical, x-ray, and lipiodol study suggests the possibility of bronchial stenosis, bronchoscopic dilatation should be immediately carried out. Holinger[4] calls attention to the fact that inflammatory bronchial stenoses, if untreated, may eventually lead to atelectasis and then to bronchiectasis. Most forms of bronchial stenoses, with the exception of the well-developed fibrostenotic type resulting from endobronchial tuberculosis, are easily dilated.

Nonopaque Foreign Bodies: Nonopaque foreign bodies of the bronchial tract, particularly in children and infants, present a most troublesome problem in diagnosis, since in many instances they are frequently overlooked as a diagnostic possibility. It is only when severe pulmonary reaction has already taken place that these patients are sent to the bronchoscopist for examination. Unfortunately, because of inadequate history and due to the fact that these foreign bodies are not visualized by the conventional x-ray films of the chest, many of these patients developing pulmonary symptoms, are at first treated for bronchitis or pneumonia.

In every child or infant seen for the first time, who is suffering from obvious pulmonary infection, a careful history obtained from the parents should include information regarding recent attacks of coughing and choking while eating or drinking. If wheezing is present, special note should be made regarding duration and mode of onset. A careful history thus obtained may prove to be

a very important guide in the diagnosis of aspirated foreign body.

Mechanics of the Bronchial Tract: In order to establish the diagnosis and to better understand the peculiar physical findings produced by nonopaque foreign bodies, it is important that one should have a basic konwledge of the mechanics of the bronchial tract during respiration. The bronchi are not rigid tubes and during inspiration they elongate and widen, conversely, on expiration they become shortened and narrowed. This constant change in the caliber of the bronchial lumen during respiration plays a major role in the production of the physical signs commonly found when foreign bodies invade the bronchial tract.[5]

As a rule, once a nonopaque foreign body, particularly those vegetal in type, becomes lodged in a bronchus, a local reaction soon follows, causing a severe edema of the bronchial mucosa which further increases the bronchial obstruction. On inspiration the bronchi being normally widened, the invasion of a bronchus by a foreign body, at first produces only a partial obstruction during this phase of respiration. Sufficient space still remains between the foreign body and the bronchial wall to allow the

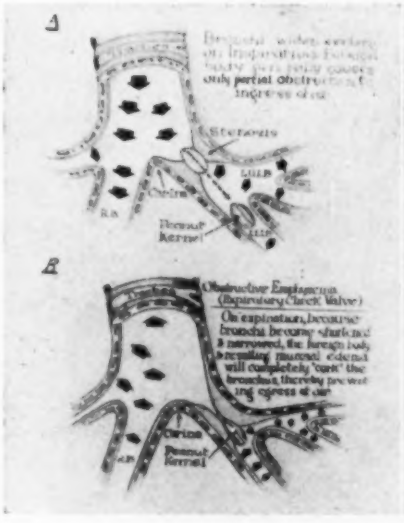
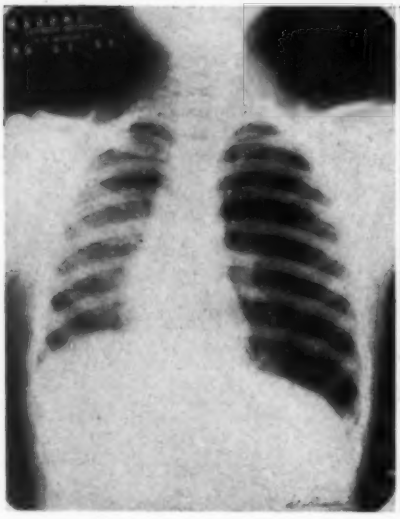


FIGURE 4

FIGURE 5

Figure 4: (A) Schematic illustration showing how a non-opaque foreign body causes only partial obstruction to the ingress of air. However, on expiration, (B) because the bronchi normally become narrowed and shortened, the foreign body and the resulting mucosal edema, will cause a complete obstruction to the egress of air. This is commonly spoken of as an expiratory check-valve.

Figure 5: If the foreign body is not soon removed, the prolonged irritation and resulting local edema will completely obstruct the bronchus, thereby preventing both the ingress and egress of air. The check-valve mechanism which was only expiratory in type, has now changed to a complete check-valve, and the state of the lung, formerly emphysematous, is now completely atelectatic.



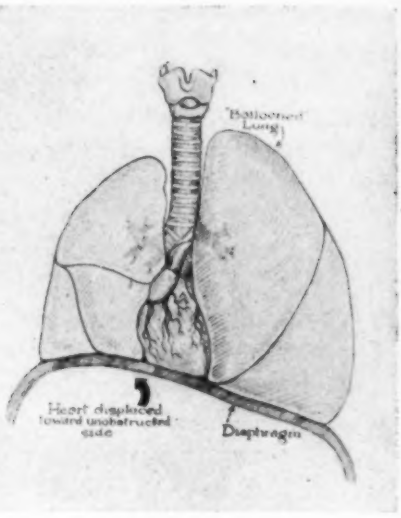
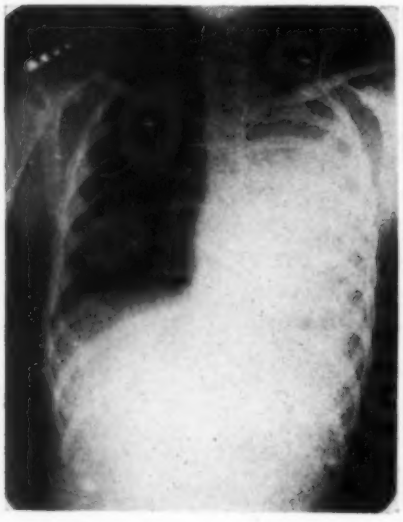


FIGURE 6 FIGURE 7

Figure 6: Radiograph of child age ten, with non-opaque foreign body in left main stem bronchus resulting in obstructive emphysema of the left lung. The mechanics involved are those of an expiratory check-valve.

Figure 7: Schematic drawing showing the appearance of the involved lung in obstructive emphysema as shown in figure number six.



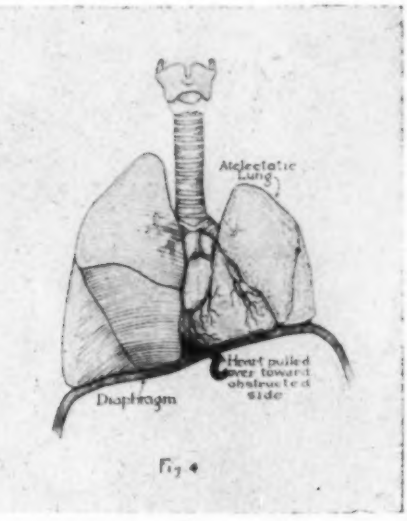


FIGURE 8 FIGURE 9

Figure 8: Radiograph of same child taken one week later showing a complete atelectasis of the left lung. The mechanics now involved are those of a complete check-valve. The bronchus is completely corked, thereby preventing both the ingress and egress of air.

Figure 9: Schematic drawing illustrating the appearance of the involved lung in atelectasis.

ingress of air. However, on expiration the situation becomes entirely reversed. The bronchi now normally narrowed, the foreign body and the resulting local mucosal edema will in varying degree obstruct the egress of air from that portion of the lung involved. This stage of expiratory obstruction results in a hyperdistension of the pulmonary segment distal to the obstructed bronchus. In other words, a condition of obstructive emphysema is in the making. The mechanics involved in producing this condition is often referred to as an expiratory check valve mechanism. If there is any delay in the removal of the foreign body during this stage of obstructive pulmonary emphysema, the increased mucosal swelling and accumulated secretions resulting from the prolonged local irritating action of the foreign body, will now completely cork the bronchus, thereby preventing both the ingress and egress of air from that portion of the lung supplied by the obstructed bronchus. The mechanism, which previously was only expiratory in type, has now changed to that of a complete check valve obstruction and the state of the lung, formerly emphysematous, is now completely atelectactic (Figs. 4 and 5).

Even during this early stage of obstructive atelectasis, the removal of the foreign body and aspiration of the retained secretions will in many instances restore the lung to its normal function. However, when atelectasis is allowed to exist for a period of time, the resulting pulmonary changes may prove to be irreversible. This last stage of complete bronchial obstruction, with

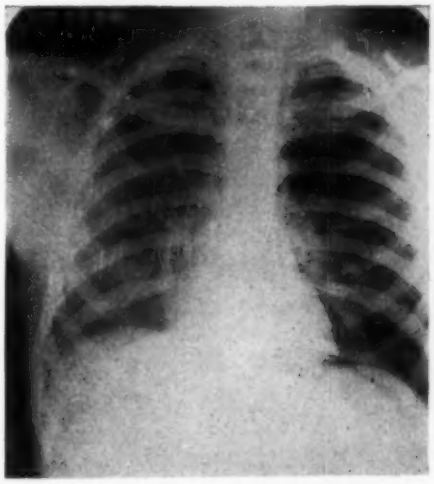


Figure 10: Radiograph of same child taken one week after the removal of the non-opaque foreign body, the rubber sac of a fountain pen, which had gone undiagnosed for a period of three weeks. In spite of the fact that obstructive atelectasis had existed for a period of several days, the child made uneventful recovery following the removal of the foreign body and aspiration of the retained secretions.

subsequent pulmonary destruction might never be reached if every pediatrician and general practitioner of medicine would strongly consider the diagnostic possibility of aspirated foreign bodies as the causative factor in both children and infants who are suffering from obvious bronchial or pulmonary infection.

The symptoms and physical signs will vary with the degree of bronchial obstruction and with the amount of interference with aeration and drainage of the subjacent portion of the lung involved. Certain types of nonopaque foreign bodies, beans in particular, which because of their chemical structure possess the property to expand or enlarge in the presence of moisture, may quickly produce a complete bronchial obstruction, resulting in a massive atelectasis within a few hours following the intrusion. Irritating vegetal bodies such as peanut kernels will usually set up a violent reaction immediately following the bronchial lodgement (Figs. 6, 7, 8, 9, 10 and 11).

Bronchoscopy and Treatment

Treatment of Lung Abscess: In the field of therapy, bronchoscopic aspiration for the treatment of suppurative diseases of the bronchial tract is now a well established procedure. By no means is it necessary to recommend all cases of lung abscess for thoracic surgery. In a series of 36 cases of early lung abscess

Figure 11: Postmortem specimen of lung from female patient age 67, showing complete obstruction of right main stem bronchus by a large piece of nut. She was first seen by a physician three weeks before her death, who diagnosed her condition as pneumonia. She eventually developed atelectasis with pulmonary suppuration. When atelectasis is allowed to remain for any great length of time, the resulting pulmonary changes may prove to be irreversible and the outcome may prove fatal.

seen by the author during the past ten years, in only 9 cases was it necessary to employ surgical intervention. The 27 cases not requiring surgery made uneventful recoveries following bronchoscopic aspiration, antibiotic and chemotherapy. It is to be made clear, however, that bronchoscopic aspiration and conservative measures should be carried out only for a period of four to six weeks. If, during that period of time no improvement has been shown, either clinically or by roentgenographic study, or if at any time during this period of conservative management there appears to be a progression of the disease, surgery is immediately advocated.

The desired factor in the bronchoscopic treatment of lung abscess is to facilitate drainage from the abscess cavity. Usually the draining bronchus leading from the cavity becomes blocked by mucosal edema, thick purulent secretions, and granulation tissue. Once the affected pulmonary segment is carefully localized by preliminary physical and x-ray examinations, the bronchoscopic procedure consists of locating and draining the involved bronchus. Whenever possible, the bronchoscopic injection of iodized oil directly into the draining bronchus and into the abscess cavity will materially aid drainage by its displacement of tenacious purulent secretions. Since the advent of penicillin, streptomycin and the sulfonamide group of drugs in the treatment of many types of pulmonary infections, this form of antibiotic and chemotherapy is also employed in combination with the treatment by bronchoscopy. In several cases treated during this past year sulfathiazole and penicillin were insufflated and instilled directly into the draining bronchus. If bacteriologic examination also reveals the presence of fusospirochetal organisms, intravenous injections of neoarsphenamine are given at weekly intervals for a period of several weeks.

In the treatment of chronic lung abscess, except as a preliminary aid to the thoracic surgeon, bronchoscopy is of small value. Cases having a well-defined, densely organized abscess wall are strictly a surgical problem.

Treatment of Bronchiectasis: The only treatment offering a complete cure in late bronchiectasis is lobectomy or total pneumonectomy. However, where surgery is contraindicated because of advanced age, multilobar involvement, and cardiac disease, routine bronchoscopic aspirations, postural drainage, and intratracheal instillation of penicillin and iodized oil offer a marked palliative relief to the patient. As an aid in the surgical treatment of bronchiectasis, bronchoscopic aspiration of purulent secretions immediately before, and occasionally during and following the operation, serves to reduce the incidence of postoperative com-

plications. This simple procedure often proves to be a life-saving measure.

Cause and Prevention of Bronchiectasis: One of the most important roles assigned to the field of bronchoscopy is in the prevention of primary or acquired bronchiectasis. The accumulated clinical, experimental, and post-mortem studies clearly indicate that intrinsic or primary bronchial dilatation is practically always due to infection of the bronchial wall with destruction of the elastic tissue framework, resulting from prolonged bronchial occlusion and atelectasis. In the majority of cases this condition is chronic and its evolution is gradual. Occasionally bronchiectasis is met with in the acute form following infectious diseases, characterized by marked bronchial irritation, as in whooping cough, influenza and measles.

Children and infants suffering from upper respiratory infection with persistent aspiration of postnasal drainage material are in constant danger of developing atelectasis and finally bronchiectasis. It is most important that if the condition is to be prevented and the chronic source of bronchial irritation removed, the upper respiratory infection should be eliminated. Also after the successful removal of aspirated foreign bodies which have caused some inflammatory reaction, patients should be kept under observation and should undergo periodic chest examinations for a period of several months.

Holinger[4] and Tucker[6] describe various stages in the development of bronchiectasis, in which, if bronchoscopic aspiration is frequently done and bronchial drainage is re-established, the existing pathologic process is still reversible. Anspach[7] calls attention to the so-called triangular shadows at the base of the lungs, which he regards as an area of localized atelectasis, where, under certain conditions, bronchiectasis may develop. To Anspach also goes the credit for calling the attention to the value of bronchoscopic aspiration in the prevention of bronchiectasis in children presenting this basal involvement.

The rapidity with which bronchiectasis develops after pulmonary atelectasis will to a great extent depend on the type and virulence of organisms present in the trapped secretions. Because of the airless state produced by the atelectasis, a state of anaerobiosis is soon established. If anaerobic organisms are present, they will multiply in great numbers and hasten the destruction of the elastic tissue framework of the bronchial structures. Smith[8] believes that fusospirochetal organisms aspirated from the oral cavity and always present in the secretions of obstructive lesions of the bronchial tract play a very important role in the development of bronchiectasis.

The diagnosis of bronchiectasis can only be definitely established by means of pneumonography. Neither the internist nor the radiologist should attempt to make a positive diagnosis unless the above procedures have been first carried out.

Postoperative Atelectasis: This development usually results from a bronchial obstruction by aspirated secretions or vomitus during or immediately following operation. The condition is more common following operations in the upper adbdominal quadrants because of decreased diaphragmatic movement in an attempt to reduce abdominal pain. Although postoperative atelectasis was clearly described as early as 1910, its frequency has been fully appreciated only in recent years. Improved x-ray technic and the many papers published on obstructive atelectasis have stimulated both internists and surgeons, with the result that this condition is now a definite entity with definite clinical and x-ray evidence.

The symptoms and the observations made of this postoperative complication usually follow a rather definite pattern. Within several hours or longer, following operation, the patient complains of dyspnea, which is usually out of all proportion to the degree of pulmonary involvement. There is a sense of discomfort over the lung involved, the pulse becomes rapid, there is sudden increase in temperature, and cyanosis becomes apparent. Contrary to general opinion, the diagnosis of this condition in its early stage is dependent more upon the physical examination than the x-ray findings. Physical findings during this early stage are usually those of obstructive emphysema.

As the bronchial obstruction increases, early signs of atelectasis begin to appear. The x-ray findings in the early cases of atelectasis may reveal little or no increase in density over the lung involved, but later the shadow may be dense or homogeneous. In the more severe cases, there is an elevation of the diaphragm on the affected side, narrowing of the intercostal spaces, and marked displacement of the heart and mediastinum.

Treatment of Postoperative Atelectasis: Postoperative atelectasis can be best treated by means of bronchoscopic aspiration, with the removal of the obstructing bronchial secretions. It is important that if an early diagnosis is to be established, the surgical intern and the surgeon be ever on the alert for the possibility of this complication. Fortunately for the patients who come to operation in the author's community, surgeons and internists are wide awake regarding this complication and lose no time in asking for bronchoscopic consultation. In the past, patients developing this unfortunate condition were treated for pneumonia, a procedure that was usually followed by a high mortality. Today practically all patients treated for postoperative

atelectasis by bronchoscopic aspirations make an uneventful recovery. In the majority of cases treated, there is a sudden drop in the temperature, pulse, and respiratory rate. Almost immediately the patient feels relieved of the dyspnea and is able to cough up with ease the remaining secretions. It is to be made clear however, that the diagnosis must be made early, for if atelectasis is allowed to exist for many hours, secondary pneumonitis and pulmonary suppuration may develop. In several well-known surgical clinics in this country, patients exhibiting signs of respiratory obstruction are immediately bronchoscoped during the operation and again before leaving the operating room. It is a standard rule in these clinics that no patient shall leave the operating room with a "wet lung" (Figs. 12 and 13).

In addition to bronchoscopic aspiration, patients developing postoperative atelectasis are administered a 5 per cent carbon dioxide-oxygen mixture for a period of five minutes at intervals of from one to two hours until respiration and pulse rate return to a normal preoperative level. Instructions are also given to have the patient frequently rotated from side to side. With this added treatment, the patient is usually able to cough up the remaining retained secretions.

Prevention of Postoperative Atelectasis: In the prevention of this complication many important details should be attended to before and during the operation. One important detail is the question of heavy preoperative sedation in patients who are known to have a chronic bronchitis or sinusitis. Many of these patients spend considerable time on awakening every morning in clearing

FIGURE 12

FIGURE 13

Figure 12: Radiograph of male patient age 54, showing a well developed postoperative atelectasis of the right lung following a gall bladder operation.

Figure 13: Radiograph of patient shown in figure 12, five days following bronchoscopic aspiration.

their nasopharynx and tracheobronchial tree of accumulated secretions aspirated during the sleeping hours. If the cough reflex is depressed by heavy sedation given the evening before and on the morning of operation, these patients are not able to clear out their airways. Before giving the morning sedation, then, these patients should be encouraged to cough and clear themselves of retained secretions.

If morphine is used postoperatively it should be given cautiously since it will tend to depress the cough reflex. Atropine and acopolamine should also be used judiciously because they tend to dry up secretions, making it impossible for the delicate ciliated epithelium to move up secretions in the larger bronchi and trachea from which they can be easily expectorated.

If there is any question whatsoever of respiratory disease a complete x-ray study of the chest should be made and, if possible, operation should be postponed if the x-ray film reveals any pulmonary disease.

Bronchoscopy in Bronchial Asthma: If possible, every case of bronchial asthma being contemplated for allergenic treatment should undergo a diagnostic bronchoscopy. To recall the words of Dr. Chevalier Jackson: "All that wheezes is not asthma." Time and again, bronchoscopic examination of patients diagnosed and treated for this condition has revealed that the underlying cause of wheezing and dyspnea was the presence of tracheal or intrabronchial disease in the form of neoplasm, bronchial stricture, bronchiectasis, localized atelectasis, endobronchial tuberculosis, various forms of bronchitis, nonopaque foreign bodies, laryngeal papillomata, and pressure on the trachea and bronchi from extrinsic causes (Figs. 14 and 15).

The bronchoscopic findings in bronchial asthma are chiefly those of mucosal edema with congestion. The amount and character of the secretion usually depend in a large measure on the severity and type of bronchial infection present. In the chronic form on bronchial asthma secondary infection is invariably present and degenerative changes of the bronchial tract with defective ciliary function are a constant finding.

The author, in the repeated bronchoscopic examination of numerous patients of all ages suffering from intractable asthma, has never observed the phenomenon of bronchial spasm to be present in the major bronchi. The constant finding of bronchial obstruction by mucosal edema with congestion has led the author to reconsider the previously accepted theory that status asthmaticus was primarily due to a spasm of the circular fibers of the bronchial wall. While the hypothesis of bronchial spasm is the one most generally advanced as the immediate cause of the

attack, it is quite probable that the factor of bronchial edema with congestion plays a very important part and that both, rather than either one alone, are involved in producing the phenomenon of an asthmatic seizure. Bronchoscopy, by means of direct inspection of the inside of the bronchial tree, affords an open door to the further study of this complex disease.

Bronchoscopy in the Treatment of Bronchial Asthma: The treatment of this condition, regardless of method or combination of therapy employed, makes up one of the gloomy chapters in the history of medicine. In many instances when the attacks

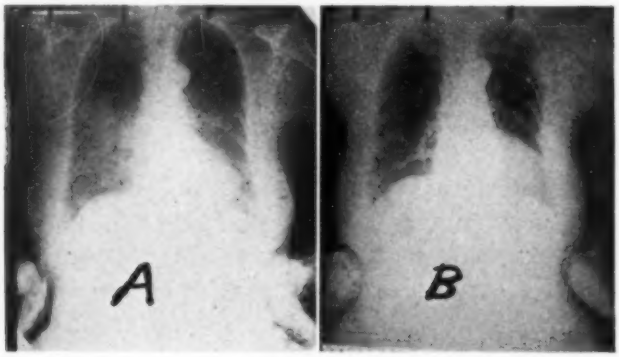


Figure 14: Radiograph of patient, (A) female age 62, who was recommended for bronchoscopic investigation, following several months of unsuccessful allergenic treatment for bronchial asthma. Bronchoscopic examination revealed a large pedunculated tumor mass arising from the posterior wall of the lower one-third of the trachea. The shifting position of the tumor mass on expiration almost completely obstructed the egress of air. The emphysematous appearance of the chest and the physical signs and symptoms present closely resembled the findings commonly seen in bronchial asthma. The tumor mass which was removed by bronchoscopic procedure in June 1942, was reported by the laboratory as an epidermoid carcinoma. (B) Radiograph taken in June 1947, reveals the disappearance of the severe emphysema as shown in figure A. This is one of the few cases still alive five years later, following the removal of a primary carcinoma of the trachea.

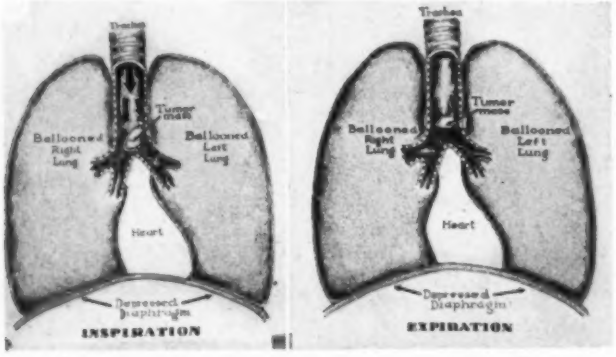


Figure 15: Schematic drawing of tumor mass of the trachea described in figure fourteen, illustrating the expiratory check-valve mechanism, which resulted in severe bilateral obstructive emphysema.

occur early in life and tend to persist and increase in severity, the patient inevitably develops chronic emphysema and sooner or later become a confirmed "asthmatic." Both the severity and duration of the seizures will determine to a great extent the rapidity with which this emphysematous state is reached.

Bronchoscopic aspiration of retained viscid secretions during an asthmatic attack which has failed to respond to the commonly accepted methods of treatment will in many instances result in the quick subsiding of the attack. Ramirez and St. George[9] believe that bronchoscopy is of undoubted value in the so-called nonallergic forms which are associated with chronic bronchitis and bronchorrhea. The benefit is derived not only from the removal of the thick mucus and the endobronchial medication but also from the vaccines prepared from the bronchoscopically aspirated secretions. Autogenous vaccines prepared in this manner, free from oral contamination, seem to be more effective than those prepared from sputum. Clerf[10] has obtained excellent palliative results following bronchoscopic aspiration of secretion in certain cases of asthma in which there was a tracheobronchitis or definite suppuration of the lung. Halliday[11] believes that the marked improvement and relief from distressing symptoms following bronchoscopic aspiration and instillation of medication render this procedure an important aid in the treatment.

In certain cases of intractable asthma with heavy, retained secretions not responding to any form of allergenic or adrenalin therapy, the author has obtained a marked symptomatic improvement following frequent intratracheal instillation of a 40 per cent iodized oil. The oil, because its specific gravity is greater than that of the bronchial secretion, displaces and floats the secretion into the larger bronchi and trachea from where it may be coughed up with ease. There is also a possibility that the free iodine being constantly liberated may serve in stimulating the sympathetic system in relieving bronchial spasticity. Iodized oil should not be employed in patients with cardiac disease, toxic goitre and severe pulmonary emphysema.

The author is in complete accord with Clerf, who states that before a patient is accepted for bronchoscopic treatment, systematic studies should be made to elicit all possible causative factors. The investigation should include a complete x-ray study of the chest, rhinologic examination, and a thorough allergic study.

Bronchoscopy in Treatment of Massive Pulmonary Hemorrhage: One of the immediate dangers of profuse hemorrhage is death by asphyxia. Emergency bronchoscopic aspiration of retained blood clots with relief of tracheal and bronchial obstruction will in many

instances save the life of the patient. As no time should be lost in instituting this procedure, the author recommends that bronchoscopic aspiration be quickly performed at the bedside. If the patient can be kept alive for a period of twenty-four hours the immediate prognosis is good. It might be necessary to bronchoscope the patient a number of times at half hour intervals. Often the syncope induced by the sudden loss of considerable blood aids in saving life by promoting thrombosis. When the author, ten years ago, first advocated bronchoscopic aspiration in the treatment of massive hemorrhage from pulmonary tuberculosis, he was severely criticized for recommending this type of procedure. The fact remains that the majority of patients who expire within twenty-four hours following massive hemorrhage, do not die from loss of blood per se, but rather from suffocation induced by tracheal and bronchial obstruction fro mclotted blood. Every patient showing impending signs of asphyxia, following pulmonary hemorrhage should be immediately bronchoscoped.

In cases of repeated hemoptysis of doubtful etiology, bronchoscopy should be employed, since not only may the bronchus to the bleeding area be identified, but it may be discovered that instead of being a pulmonary lesion the hemorrhage originates from a tumor mass or an ulcer in the trachea or large bronchus, and the lesion so situated that it can be treated locally.

Endobronchial Tuberculosis: Bronchoscopy has opened up a relatively new and important field in the diagnosis and treatment of tracheobronchial tuberculosis. The inexplicably bad results of collapse therapy in the past were no doubt due in part to the presence of bronchial tuberculosis, which was frequently overlooked. The importance of this condition is best illustrated by the fact that today it is a standard procedure with a great many chest physicians that all patients being contemplated for any form of collapse therapy must first undergo a diagnostic bronchoscopy to rule out tracheobronchial involvement. If the bronchial tract is involved, postponement of operation is preferred and bronchoscopic treatment consisting of topical application of a 30 per cent silver nitrate solution is immediately instituted. If in spite of repeated treatments, a well developed fibro-stenosis develops, which is soon followed by atelectasis, no time should be lost with attempts in dilatation, since this procedure is usually ineffective, and surgical intervention should be advised. A recent survey of the literature dealing with this serious complication reveals that a number of cases have already been successfully treated by means of pulmonary resection. (Fig. 16).

The symptoms of bronchial tuberculosis are chiefly those of partial bronchial obstruction. In some instances the obstruction

becomes complete and a state of atelectasis soon develops. This complication of pulmonary tuberculosis should always be suspected in patients who suddenly develop bronchial wheezing, elevation of temperature, decrease in the amount of expectoration, hemotysis and signs of increasing dyspnea. Auscultation usually reveal the presence of harsh rhonchi along the sternal border on the affected side. In a small group of cases with positive sputum and hemoptysis in which there is no demonstrable x-ray evidence of pulmonary disease, bronchoscopic examination often reveals the presence of tuberculous bronchial ulceration.

Many tuberculosis institutions throughout the country, realizing the importance of bronchoscopy as an aid in the diagnosis and treatment of this serious complication of pulmonary tuberculosis, have now added a bronchoscopic clinic as an integral part of the surgical department.

Bronchoscopy as an Aid to Thoracic Surgery: The rapid progress already made to date in the field of thoracic surgery has been in no small way due to the aid of the bronchoscopist, who by careful endoscopic examination has aided in the correct localization of pulmonary lesions. Probably the greatest aid of bronchoscopy to the thoracic surgeon has been in the early diagnosis of primary carcinoma of the lung. In the diagnosis of tracheobronchial tuberculosis, the bronchoscopist has been of great aid to the surgeon in calling his attention to the fact that any form of collapse therapy will result in failure unless the endobronchial lesion is also bronchoscopically treated. In certain instances bronchoscopic aspiration before, during, and following thoracic opera-

Figure 16: Endobronchial tuberculosis. Fibrostenosis of left main stem bronchus, resulting in complete atelectasis of left lung. This type of lesion does not respond to dilatation and no time should be lost with attempting this type of procedure. Pulmonary resection is advised for this type of case.

tions has served to reduce postoperative mortality. The advances still to be made in the field of thoracic surgery will no doubt depend in part on further bronchoscopic research on the behavior of the normal and diseased bronchi.

SUMMARY

In this brief review of the role of bronchoscopy in clinical medicine and surgery, the author has attempted to emphasize the following facts:

1) Aside from the well-known procedure for the removal of aspirated foreign bodies, bronchoscopy has come to play an important role in the diagnosis and treatment of diseases of the lungs.

2) There are many conditions of the lower respiratory tract that often remain undiagnosed because of lack of bronchoscopic study.

3) In the diagnosis of primary carcinoma of the lung, bronchoscopic examination is by far the most important diagnostic procedure available.

4) Close cooperation between the internist, radiologist, and bronchoscopist will in many instances facilitate the diagnosis of obscure conditions of the lung.

5) Many suppurative conditions of the lungs if diagnosed early, will respond to bronchoscopic and medical management.

6) Bronchoscopy is of aid in the removal of bronchial obstruction, thus preventing the development of atelectasis and subsequent pulmonary destruction.

7) Bronchoscopy is of aid to the thoracic surgeon in the localization of pulmonary lesions.

1301 Union Street.

RESUMEN

En esta breve revista de la función de la broncoscopia en la medicina y cirugía clínicas, ha tratado el autor de recalcar los hechos siguientes:

1) Además del procedimiento bien conocido de extraer cuerpos extraños aspirados, la broncoscopia ha llegado a jugar un papel importante en el diagnóstico y tratamiento de las enfermedades de los pulmones.

2) Hay muchos estados del aparato respiratorio inferior que quedan a menudo sin diagnóstico por falta de estudio broncoscópico.

3) El examen broncoscópico es, con mucho, el procedimiento más importante a nuestra disposición para diagnosticar el carcinoma primitivo del pulmón.

4) La íntima cooperación entre el internista, el radiólogo y el broncoscopista facilitará en muchos casos el diagnóstico de condiciones oscuras del pulmón.

5) Muchos estados de supuración de los pulmones, si se diagnostican oportunamente, responden al tratamiento broncoscópico y médico.

6) La broncoscopia ayuda a eliminar obstrucciones bronquiales, evitando así el desarrollo de atelectasia y de la destrucción pulmonar subsiguiente.

7) La broncoscopia ayuda al cirujano torácico a localizar las lesiones pulmonares.

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A Demand Valve for the Administration of Aerosols of Penicillin and Streptomycin*

MAURICE S. SEGAL, M.D., F.C.C.P.,** LEON LEVINSON, M.D.,*** and JOHN F. BEAKEY, M.D.****

Boston, Massachusetts

The efficacy of penicillin and streptomycin aerosols[1-4] administered via the pulmonary route for the management of bronchopulmonary sepsis has become well established. Such therapy has been employed successfully in patients with the following conditions: acute and chronic infectious sinusitis;[5-7] chronic sinusitis associated with bronchiectasis;[5,6] acute bacterial infections,[3,7,8] such as laryngitis, tracheobronchitis and laryngotracheobronchial edema (particularly in patients who have had a tracheotomy); bronchitis and bronchial asthma[2,3,7,9] associated with definite bacterial infections; bronchiectasis;[2,3,4,10,11,12] lung abscess;[6,10] pulmonary infections[3,6,13] due to the streptococcus, staphylococcus or penicillin-susceptible strains of Klebsiella pneumoniae; pulmonary infections secondary to pulmonary emphysema,[6,14,15] lung cysts or infarcts; unresolved pneumonic infections; pneumonitis secondary to foreign bodies and tubercular infections in the lung;[16] staphylococcal bronchopulmonary infections associated with pancreatic disease in children;[17] and it also has been proven a valuable pre- and postoperative medication in surgery of the upper and lower respiratory tract.

Having once determined that antibiotic aerosol therapy is indicated, the technique best suited to the situation is selected. Therapeutic aerosols can be administered by the simple rebreathing technique[2,3] which may be modified according to the age of the patient and the nature of the illness. For patients with obstructive disease of the respiratory tract, streams of helium and oxygen may be used instead of oxygen. The aerosol may be directed into tents, hoods, masks or tracheotomy openings. For patients with paranasal sinus infections, the aerosol may be employed nasally with or without alternating positive and negative pressure.[5,10] Finally, if indicated, other drugs may be substituted, alternated or combined with penicillin or streptomycin.

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*From the First and Third Medical Services, Boston City Hospital, and the Department of Medicine, Tufts College Medical School.

**Assistant professor of medicine, Tufts College Medical School; Director, Department of Inhalational Therapy, Boston City Hospital.

***Assistant in medicine, Tufts College Medical School.

****Assistant resident in medicine, Department of Inhalational Therapy, Boston City Hospital.

Numerous factors are involved in the degree of retention of aerosols in the respiratory tract and of their diffusion into the blood stream. The following are the more important ones: (1) particle size, which depends essentially on the type of nebulizer and technique employed; (2) point of ultimate deposition of the particles which depends upon impingement, settling and diffusion by Brownian movements; (3) pattern of the ventilatory curve, particularly the respiratory rate, tidal air and the degree of bronchospasm; (4) aerodynamic factors, such as turbulence and stream line flow; and (5) factors which may influence the size and life of the particles themselves, namely stability and surface tension of the aerosols.

The selection of the proper nebulizer for the production of therapeutic aerosols is of primary importance. If very large particles are inhaled, they will be deposited in the throat and upper respiratory tract by impingement or settling. On the other hand, very small particles will penetrate to the alveoli by Brownian movement, but they will largely be exhaled. Maximum alveolar deposition occurs at some intermediary size. Nebulizers should be constructed with a suitable baffle to remove the larger particles and at the same time insure a uniform delivery of the small ones, which should preferably have the mean radius between 0.5 to 2 micra.

Bryson[20,21] made a critical study of the mass radius distribution of particles from two commercial nebulizers, the Vaponefrin model and the DeVilbiss No. 40. Examination of the weight - radius - distribution curve, calculated from his data on the materials delivered by these two nebulizers shows that, for all practical purposes, the mass of the particles is contained in those with radii of 0.5 or more micra. Of the two nebulizers, the Vaponefrin model appears more efficient, for it produces considerably more mass of the particles contained in the 1.0 to 1.5 micra range, and even more in the 0.7 to 1.5 micra range. Generally, a more uniform production of particles in the 0.5 to 2.0 micra range is assured with the use of the baffle plate. The quantity of the material in the very smallest particles delivered by this nebulizer is essentially negligible. Furthermore, the Vaponefrin model produces considerably less scattered large particles. This "rain" is apt to occur with most nebulizers because of their imperfect construction and it is as much of a loss as the residual liquid which adheres to the walls of the nebulizer.

We have found the Vaponefrin* nebulizer most satisfactory for the production of therapeutic aerosols. It is a glass or plastic

*Vaponefrin Co., 6812 Market Street, Upper Darby, Pennsylvania.

vessel containing a special type of baffle unit and produces an exceedingly fine, voluminous mist or smoke screen, which is further refined in particle size by the baffling action of the walls of the vessel. The majority of the particles average 1 micron in radius and are thus capable of penetrating the alveoli. The nebulizer has an outlet orifice, at least as large as the diameter of the human trachea, from which the patient inspires the vapor or mist. It also has an auxiliary air jet orifice which is considerably smaller. A nipple for attachment of a hand bulb or rubber tube from an oxygen or compressed air tank provides power for the nebulizing unit (Fig. 1).

When appreciable quantities (1 to 4 cc.) of solutions of antibiotic aerosols are used, it is customary to operate the apparatus with a flow of oxygen at 5 to 7 liters per minute. A continuous flow of oxygen will obviously produce a continuous flow of aerosol. If the patient breathes through his mouth, with his lips pursed around the outlet orifice of the nebulizer, he will draw into his respiratory tract that amount of aerosol which is produced during inspiration; that produced during expiration will be wasted.

To compensate for this loss of aerosol, it has become customary to insert in the oxygen supply line a T or Y tube with its side arm open to the outside air, or a simple vent in the rubber tubing close to the nebulizer. When the side-arm or vent is occluded by the finger tip, oxygen is forced through the nebulizer and aerosol is produced; when the finger is released, oxygen escapes without formation of aerosol. This technique is satisfactory for ambulatory and cooperative patients who must be instructed in the exact timing necessary to prevent aerosol waste without stopping the production of aerosol entirely. However, if the patient is critically

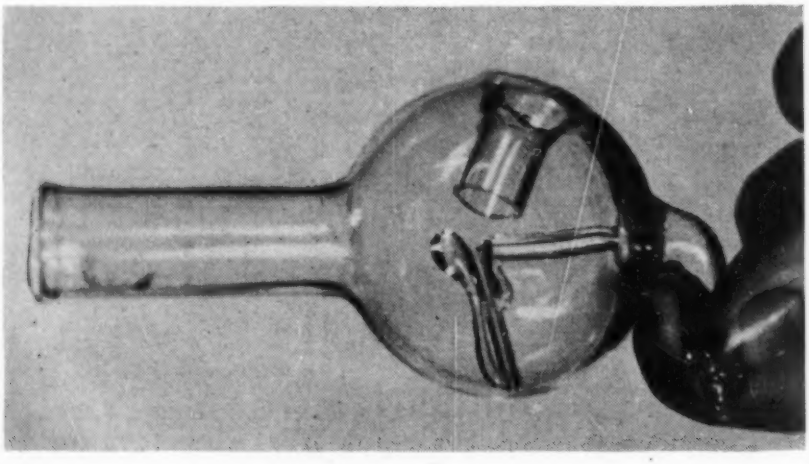

FIGURE 1

ill with bronchopulmonary sepsis, and is too toxic, disoriented or uncooperative to learn this occlusive technique, an attendant must be present constantly during the administration of penicillin or streptomycin aerosol. This procedure is not too efficient for such patients, since the administration of 1 cc. of solution via the nebulizer requires fifteen minutes or more, and the attendant can only approximate the exact timing of inspiration and expiration.

Eckman, Rumsey and Barach[22] have previously described a demand apparatus for the delivery of aerosols during inspiration only. It operates by virtue of the pressure differential set up within a Vaponefrin nebulizer with respiration, using that change to alter the tension in a coil spring which, via a thrust bearing, actuates a shut-off valve leading from the oxygen supply. We have found this apparatus too expensive and complex for routine hospital use.

A simpler, more useful demand valve (Fig. 2) has been devised to accomplish automatically the production of antibiotic aerosols during the inspiratory phase of respiration only. It improves the efficiency of this mode of therapy by increasing the actual amounts of antibiotic inspired per dose placed in the nebulizer; it reduces the cost of such therapy; and, at the same time, it obviates the instruction of patients and personnel in the Y tube technique.

The operation of this demand valve is based upon the fact that the carburetor orifice of the Vaponefrin nebulizer is considerably smaller than the outlet orifice. Therefore, inspiration with the lips pursed around the outlet orifice will set up a negative pressure (a pressure less than atmospheric) within the chamber of the nebulizer. Oxygen flowing into the nebulizer in the ranges

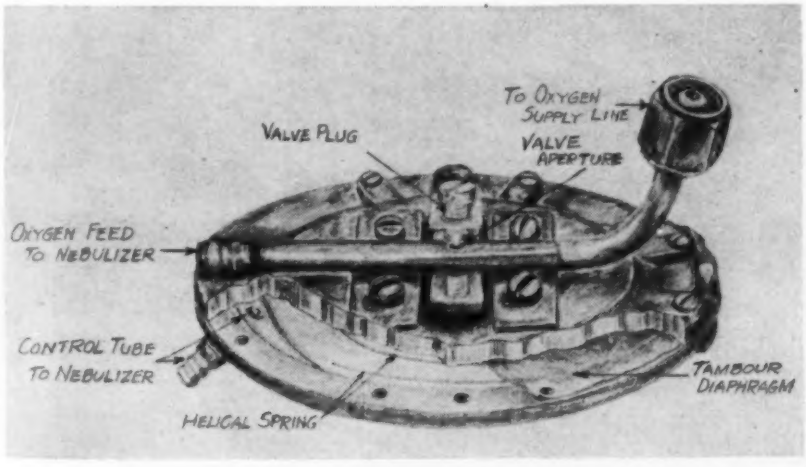

FIGURE 2

employed clinically has no effect on this negative pressure. Conversely, a positive pressure will develop during expiration.

The valve* consists essentially of a tambour type of rubber diaphragm (Fig. 2). The tambour cavity is connected by rubber tubing either to a special side-arm in the nebulizer or to any portion of the nebulizer apparatus within which a pressure differential is set up during respiration. Considerable oscillation of the diaphragm is produced by inspiration and expiration with lips pursed about the outlet orifice of the nebulizer. A short length of metal tubing, which has a side orifice and through which oxygen is passed from the regulator to the nebulizer, is mounted above the tambour diaphragm. A screw type of valve plug, which fits the orifice in the side of the metal tubing, is attached to the rubber diaphragm by an L shaped arm. Below the diaphragm, and within the tambour cavity, is a helical phosphor-bronze spring, which presses upward against the diaphragm and carries the weight of the valve plug. The purpose of this spring is to make certain that the valve will always be open (and thus that aerosols will not be produced), unless the patient, by inspiration, produces sufficient negative pressure to overcome it and close the valve. The exact degree of negative pressure necessary to close the valve is regulated by changes in the tension of the spring and by adjustment of the screw valve plug. The valve may easily be adjusted to operate on a pressure of less than 2 cm. of water.

*Manufactured by J. H. Emerson Co., 22 Cottage Park Avenue, Cambridge, Massachusetts.

FIGURE 3

This demand valve, with the helical spring, operates upon a true demand principle, producing aerosols only when a negative pressure exists within the nebulizer. We have[6,13,18] also employed valves without the spring, which in actual operation produces aerosol at all times except during active expiration (i.e. the positive pressure phase).

This spring demand valve, unlike the Emerson demand valve previously described[6,13,18] is readily adaptable to any modification of the basic Vaponefrin nebulizer. The simplest apparatus is the nebulizer with a side-arm mounted in the nebulizing chamber, to which the valve is attached, with or without the large glass rebreathing bulb (Fig. 3). For patients too debilitated for such a device, the nebulizer can be mounted in an Emerson plastic face mask* which has a head strap to hold it in place (Fig. 4). Further modifications of design are innumerable. The only basic requirement is that the apparatus somewhere contain a chamber in which a pressure differential will be developed during respiration, so that the valve may be actuated and aerosols produced. In cases where respiration is so shallow that the valve does not close with inspiration, addition of 5 per cent carbon dioxide to the oxygen can produce the desired depth of respiration.

The exact efficiency of this demand valve for the administration of penicillin aerosol is difficult to determine. Various authors have made studies of the penicillin levels in the blood after administration of penicillin aerosol, but these levels do not neces-

*Also obtainable from J. H. Emerson Co.

FIGURE 4

sarily indicate the therapeutic value of the medication, since, in contrast to intramuscular or intravenous administration of penicillin, aerosol administration is essentially a form of topical administration to the mucosal surfaces of the respiratory tract. Aerosol therapy affords a more intimate contact of the antibiotic agent with the infecting organisms in respiratory infections. Moreover, the blood levels obtained after aerosolization of penicillin are dependent upon many factors other than the dosage

TABLE 1

Penicillin in Serum after Inhalation of 100,000 Units of Crystalline Potassium Penicillin in 1 cc. of Saline Using the O.E.M. Mask Connected Directly to the Oxygen Tank Regulator.

Hours After Dose	Number of Sera Tested	Per cent Positive Sera*	Penicillin, Units Per C.C. of Serum				Ave. Units Urinary Excretion
			0.04	0.04	0.08	0.16	
½	9	67%	3	6			
1	8	50%	4	4			671 (10)**
2	9	0%	9				469 (10)
24							392 (6)
TOTAL	26	39%	16	10			1,532
							Measurable Wastage—Mouth Wash.... 1,487 (10)
							Apparatus Wash 18,670 (9)
							Total..... 20,557

*0.039 unit per cubic centimeter or more.
**number of tests done for average result.

TABLE 2

Penicillin in Serum after Inhalation of 100,000 Units of Crystalline Potassium Penicillin in 1 cc. of Saline Using the Two Liter Glass Rebreathing Bulb with the Negative Pressure Demand Valve.

Hours After Dose	Number of Sera Tested	Per cent Positive Sera*	Penicillin, Units Per C.C. of Serum				Ave. Units Urinary Excretion
			0.04	0.04	0.08	0.16	
½	6	83%	1	2	3		
1	6	67%	2	4			1,119 (6)**
2	6	0%	6				938 (5)
24							44 (3)
TOTAL	18	50%	9	6	3		2,101
							Measurable Wastage—Mouth Wash 955 (2)

*0.039 unit per cubic centimeter or more.
**number of tests done for average result.

or apparatus used. We[3,16,18] have had excellent clinical results with penicillin aerosol treatment in some cases of lobar pneumonia and suppurative lung disease where the levels of penicillin in the blood were below what we now know are minimum therapeutic levels.

The antibiotic levels obtained in the sputum may be more indicative of successful aerosol therapy than the blood levels. We have, nevertheless, determined the levels of penicillin* in the

*This study was aided in part by a grant from the Commercial Solvent Corporation.

TABLE 3

Penicillin in Serum after Inhalation of 100,000 Units of Crystalline Potassium Penicillin in 1 cc. of Saline Using the O.E.M. Mask with the Springless Positive Pressure Demand Valve.

Hours After Dose	Number of Sera Tested	Per cent Positive Sera*	Penicillin, Units Per C.C. of Serum				Ave. Units Urinary Excretion
			0.04	0.04	0.08	0.16	
½	7	86%	1	4	2		
1	7	57%	3	4			883 (7)**
2	6	0%	6				709 (7)
24							936 (1)
TOTAL	20	50%	10	8	2		2,528
							Measurable Wastage—Mouth Wash 1,117 (4)

*0.039 unit per cubic centimeter or more.
**number of tests done for average result.

TABLE 4

Penicillin in Serum after Inhalation of 100,000 Units of Crystalline Potassium Penicillin in 1 cc. of Saline Using the Emerson Mask with the Negative Pressure Demand Valve.

Hours After Dose	Number of Sera Tested	Per cent Positive Sera*	Penicillin, Units Per C.C. of Serum				Ave. Units Urinary Excretion
			0.04	0.04	0.08	0.16	
½	6	100%	0	2	2	2	
1	6	67%	2	2	2		1,166 (6)**
2	6	17%	5	1			2,046 (5)
24							522 (2)
TOTAL	18	61%	7	5	4	2	4,234
							Measurable Wastage—Mouth Wash 7,156 (4)

*0.039 unit per cubic centimeter or more.
**number of tests done for average result.

serum of normal individuals following the administration of penicillin aerosols via the demand valve and face mask, and via the demand valve and nebulizer with rebreathing bulb.

In Tables 1, 2, 3 and 4 we have presented these findings; the amounts of penicillin recovered from mouth rinsings and from the apparatus after use. The mouth was rinsed with 50 cc. of saline and the apparatus with 1 cc. of saline following each treatment. This technique was chosen to approximate more closely the one that would be carried out in the wards of the hospital. For comparison, average values with other apparatus are included. Study of the tables will reveal that this apparatus is certainly as efficacious as other types of apparatus.

SUMMARY

A mechanical demand valve for use with nebulizer and face mask for the automatic administration of antibiotic aerosols in inspiration, without waste of material, is presented. It is inexpensive, simple, yet sturdy in construction and operation and yields penicillin blood levels which are comparable to those of other methods that require training of patients and personnel.

RESUMEN

Se describe una válvula de demanda mecánica que se emplea con nebulizadores y máscaras en la administración automática de aerosoles antibióticos durante la fase inspiratoria, sin desperdicio de la substancia. Aunque barata y sencilla, es de fuerte construcción y operación y produce niveles sanguíneos de penicilina comparables a los que se obtienen con otros métodos que requieren el adiestramiento de los pacientes y del personal.

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Tumors of the Anterior Mediastinum
A Report of Six Cases

MILTON S. LLOYD, M.D., F.C.C.P.
New York, New York

We are indebted to Heuer and Andrus[1] for the most complete survey of mediastinal tumors in the recent literature. Hedblom[2] and Harrington[3] in contributions to the subject pointed out the usual complications and the difficulties in handling such cases when these complications occur. In referring to one of his cases Lilienthal[4] raises the question as to whether, under certain circumstances, drainage of cystic formations accompanying some of these tumors—particularly the teratomata—is justifiable.

"Early diagnosis" is the urgent demand of modern medicine. In the realm of diseases of the chest we are fortunate that the presence of air, a contrast medium and low cost x-ray examinations combine to offer a presently available solution in at least one field of endeavor.

Six cases of tumor of the mediastinum are reported below: one lipoma, one lymphangioma, one undifferentiated and three teratomata. The presence of everyone of them was signalized by a complication which made its management difficult or even endangered or cost life. With the exception of Case III, a new born infant, they illustrate the long "silent period" which characterizes them. They also illustrate the possibility that the complication may completely obscure the underlying disease or may so increase in importance as to require a shift in strategy by which the complication is treated first and independently (Cases V and VI). In Case V, drainage of an infected cyst was necessary as a life saving measure, although it was recognized that a chest wall sinus would impair the prognosis in a subsequent operation. In Case VI, months of symptomatic and supportive treatment, including the drainage of an infected cyst, were necessary to assist the patient in recovery from a chronic empyema before the removal of her mediastinal tumor could be considered.

In both cases, the procedure in retrospect appears to have proven its justification. In Case V, the patient's status was changed by drainage from a bad to a good surgical risk. In Case VI, a tumor, the bulk of which occupied more than half the width of the thoracic cavity, shrank to such a degree after drainage that it was completely covered by the gladiolus sterni. It is even questionable whether drainage at an earlier time might not have

prevented adhesions to the right mediastinal pleura and have made possible the removal of the tumor at the final operation without rupture of that membrane.

The answer to Lilienthal's question would, therefore, appear to be that under certain circumstances, drainage of a teratomatous cyst is not only practicable but necessary.

The rupture of a cyst does not, of itself, prolong recovery. Compare Case VI, requiring five operations and more than one year of hospitalization, with Case IV, requiring one operation and twenty-four hospital days. The point of paramount importance is to avoid infection by aspiration when necessary or, better still, by operation before aspiration is necessary.

In the discovery of early tuberculosis, x-ray is gradually replacing physical examination as a diagnostic method. Mass surveys of apparently healthy persons and the prompt use of the x-ray for minor symptoms is the accepted procedure in the field of tuberculosis. Not enough prominence has been given to the application of the same concept to the realm of other diseases. The narration of experiences in the following case reports is an indication for its wider and earlier use. In all but Case III, the tumor could have been discovered months, or even years earlier by x-ray, but only by x-ray, and, comparatively speaking, early diagnosis is of greater importance in nontuberculous than in tuberculous disease involving the organs of the chest. This is the great lesson to be learned from a study of these reports.

In addition, two new agents were used in the treatment of a pyocyaneus infection. Streptomycin was found to be very effective in a concentration of 10,000 units per cc. where the entire infected area could be reached by a single application. The organism appeared, however, within a period of twenty-four hours, to acquire a resistance to the agent and was thenceforth unaffected by concentrations up to ten times that strength. This was impressively demonstrated by its use in two adjoining wounds, one of which communicated with sinuses which could not be fully explored. This wound continued to produce an abundant growth with typical green coloration while the other immediately became sterile and closed rapidly (Case VI). Parachlorophenol[5] had an entirely different effect. It inhibited growth of pyocyaneus but did not destroy the organism. On culture, after contact with this drug, the typical color used for identification was not present, although the morphology remained unchanged (Case VI). In spite of this, the pathogenic properties of this usually stubborn bacillus disappeared. There was prompt and continuous progress toward recovery. The use of this chemical would appear to deserve the credit for saving this patient's life.

CASE I, FIG. 1 CASE I, FIG. 2 CASE I, FIG. 3

Case I, Fig. 1: H.D., W.F., age 56. Posteroanterior view of the chest showing a large sharply demarcated mass in the right cardiophrenic angle.—*Case I, Fig. 2:* A lateral projection of the chest locates the mass in the anterior mediastinum. *Case I, Fig. 3:* Posteroanterior view of chest seven months after operation. Note complete re-expansion of lung. Presenting symptoms had been absent since discharge.

There was no discernable systemic or local reaction in the body tissues to the drug, which was used both as an irrigation for the pleural cavity and by external application to the skin and wound in concentrations up to one per cent. Pyocyaneus did not appear to acquire resistance to parachlorophenol during a period of three weeks' continuous use.

CASE REPORTS

Case I: H. D., white female, age 56 years. She was admitted to hospital on October 13, 1944, complaining of dyspnoea and swelling of the feet and legs. She had been treated for "heart disease" for three years. In an x-ray survey, a tumor mass had been noted occupying the right cardiophrenic angle. Lateral views localized this mass in the anterior chest (Figs. 1 and 2).

Physical examination revealed shortness of breath on exertion and pitting edema of the ankles. Other findings were unimportant.

Bronchoscopic examination was carried out on October 17. No evidence of tumor within the bronchi was seen. A diagnosis of probable lipoma of the anterior mediastinum was made.

Thoracotomy was performed on October 19, under intratracheal gas-oxygen and ether anesthesia, through an antero-lateral approach in the fifth intercostal space. Short segments of the sixth and seventh ribs were resected posterior to the angle to improve exposure. A large lipoma (size of a small grapefruit) was found in the cardiophrenic angle compressing the inferior vena cava and the right atrium of the heart. It was freed with little difficulty and found to arise by a long pedicle from the pericardial fat. The tumor was removed by severing the pedicle between clamps. The chest was closed in layers without drainage. Re-expansion of the lung was effected by positive intrabronchial pressure and aspiration of the air from the pleural cavity through a needle.

Recovery was uneventful. A blood-tinged effusion was removed on two occasions. Fifty thousand units of penicillin were instilled into the pleural cavity at each aspiration. The patient was discharged on November 9. The presenting signs and symptoms had disappeared and she has since remained well (Fig. 3).

The pathologist reported two masses of fatty tissue, one 13x7x5 cms. and the other 5x3 cms. in dimension. Microscopic examination revealed fatty tissue only.

Case II: R. M., white, 4 years old. Admitted to hospital May 18, 1937, with a history of pain in right upper quadrant of four days' duration following a blow received while at play. There was a past history of breech presentation at birth but otherwise normal infancy.

On physical examination there was limited respiratory movement on the right side with dullness and diminished breath sounds over the lower half of the right lung. Temperature range was from 100° to 101° X-ray film of chest (Figs. 1 and 2) showed a large, well defined mass occupying the right lower anterior chest. Other laboratory data were without significance. A right pneumothorax was induced on May 26, which demonstrated that the mass was extrapulmonary and attached at the mediastinal side. Examination by thoracoscopy on June 4, showed the mass to be about the size of a grapefruit, firm, covered by a smooth membrane resembling pleura and exhibiting many blood vessels of

CASE II, FIG. 1 CASE II, FIG. 2 CASE II, FIG. 3

Case II, Fig. 1: R.M., W.M., age 4. Posteroanterior projection showing a mass occupying the lower half of the right pleural cavity.—*Case II, Fig. 2:* A lateral projection localizes this mass in the anterior mediastinum.—*Case II, Fig. 3:* Posteroanterior view of chest one year postoperative at which time the child was growing normally. (Photograph reversed in reproduction).

moderate size and a number of translucent irregularities suggesting cyst formation.

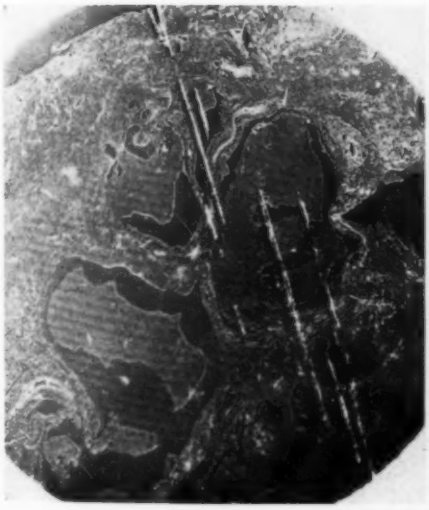
Operation was carried out on June 10, under gas-oxygen and ether anesthesia. Incision was made through an anterolateral approach in the 4th intercostal space. Adequate exposure was obtained without removal or section of ribs. The growth was attached to the pericardium, lung and diaphragm by partially organized adhesions. After separating these adhesions the base was found to arise from the wall of the inferior vena cava. This attachment could not be separated without opening the vein and the mass was cut away without much loss of blood. A No. 22 F. catheter was inserted in the 8th posterior intercostal space for drainage and the chest wall closed in layers.

Postoperative course was marked by a temperature rise from 101° to 103°. There was considerable drainage containing blood and debris which, however, remained sterile. The drainage tube was removed june 28.

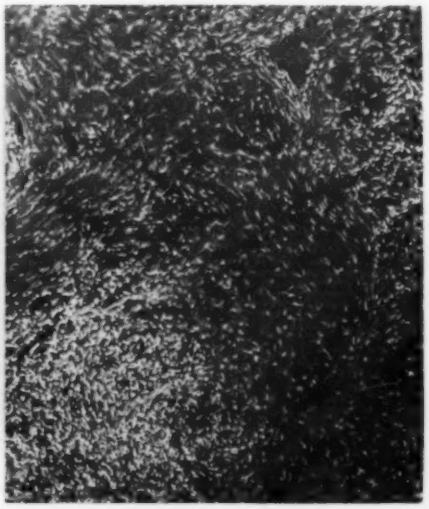
The patient received deep x-ray therapy postoperatively, a total of 3050 r units being administered by Dr. M. B. Radding.

He was discharged on September 5, 1937. A follow-up x-ray film (Fig. 3) taken one year after admission to hospital, on May 14, 1938, showed a chest of normal appearance. His development has since been normal.

Pathological report was as follows: "Specimen received in formalin in three pieces, represents an irregularly shaped mass, aggregating to the size of a tangerine. Within this mass an area, composed by smaller and larger cysts, is present, the size of a large pea. However, the greatest portion of the tumor is composed of a firm tissue which is hard elastic and on its cut surface, grayish-white, intermingled with pin-point orange-yellow areas and scattered small pin-point hemorrhages. Within this tissue there are small lumena, about 1 mm. in diameter, present.



CASE II, FIG. 4



CASE II, FIG. 5

Case II, Fig. 4: Section of tumor—low power, showing large sinuses with coagulated serous content.—*Case II, Fig. 5:* High power section of tumor showing connective tissue with scattered smooth muscle cells, giant cells and phagocytes.

Part of the tissue is surrounded by capsule. One particle of the tumor is hemorrhagic and fleshy.

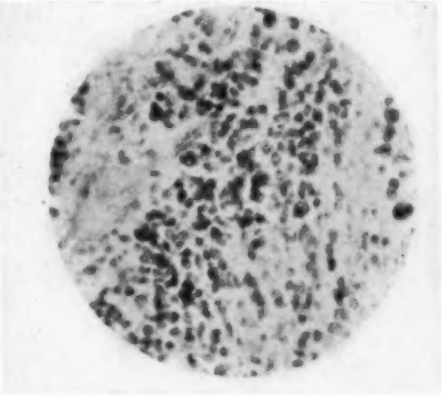
"Section consists chiefly of dense connective tissue with scattered bundles of smooth muscle fibers, which apparently belong to the wall of large sinuses. These sinuses are filled with hemogeneous fluid. There are many such large dilated sinuses. Within the stroma, there are numerous nodules, varying in size, which are formed by granulation tissue including many giant cells. These giant cells surround cholesterol crystals and many lipophages. There are several fragments with congested lung tissue with interalveolar and interseptal hemorrhages. The pleura is thickened and shows numerous newly formed blood vessels.

Diagnosis: Lymphangioma with chronic inflammation" (Figs. 4 and 5).

Case III: Baby Boy K., W. M., new born. Normal delivery. As soon as delivery was completed, patient exhibited obstructive type respiration with cyanosis and suprasternal and epigastric retraction. Bronchoscopy was performed two hours after birth with notation as follows:

"New born infant's bronchoscope was passed. A small amount of secretion was present. The carina was displaced to the left and the right wall of the trachea and right stem bronchus bulged inward causing obstruction to breathing (Fig. 1). With the bronchoscope in situ the child's condition was good but as soon as the bronchoscope was removed, dys-

CASE III, FIG. 1



CASE III, FIG. 3

CASE III, FIG. 2

Case III, Fig. 1: Baby Boy K., W.M., new born. Bronchoscopic view showing compression of the right stem bronchus by an external mass.

Case III, Fig. 2: Anteroposterior projection showing sharply defined density in upper right chest cavity.

Case III, Fig. 3: High power section of tumor showing "undifferentiated" cells in a fibrous stroma.

pnea and cyanosis returned. This is apparently caused by a mass in the right chest cavity. Patient was sent to the x-ray department for examination."

The x-ray film (Fig. 2), revealed a density in the upper right pleural cavity with sharply delineated lower border at the level of the third anterior rib. Within this shadow appeared two or three smaller areas of increased radiopacity. The heart was in normal position. The left lung was clear. The findings were interpreted as "Probable teratoma of the mediastinum extending into the right pleural cavity."

The infant died two hours later. An necropsy was performed with the following findings: "Tumor: 5-6 cm. wide, in right upper chest, base attached to soft tissue around trachea and esophagus. Tumor is solid, grayish-red on section.

Microscopic Examination (Fig. 3). Several areas are sectioned and show fine fibrous stroma, masses of cells suggestive of lymphoid tissue, and a few areas suggestive of brain tissue. There are also numerous areas of oval and polyhedral cells, quite undifferentiated. Organ elements have not been found in 7-8 areas sectioned. The structure is that of an undifferentiated embryonal tumor."

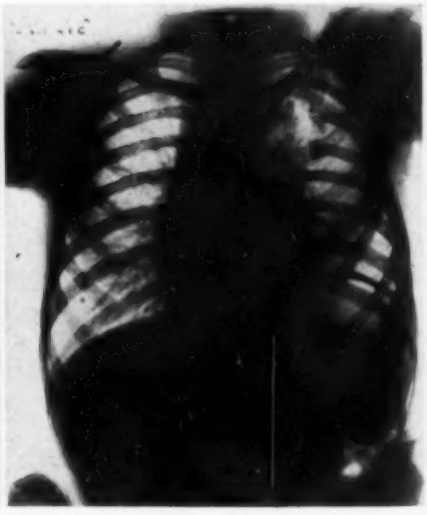
(The base of this tumor occupied the entire depth of the thoracic cavity from the sternum to the esophagus. The posterior mediastinum was not involved and there was no connection with any of the nerve trunks or ganglia. It is therefore classified as a tumor of the anterior mediastinum. There was no connection with the great vessels or other anatomical impediment to its removal. It was proposed at the time to remove the obstruction to breathing by partially marsupializing the mass, anchoring it to the ribs and retaining a covering of skin. The operation would have been completed at a second stage after feeding and other problems incidental to the new born had been solved. Unfortunately the patient died before parental consent was obtained.)

Case IV: H. N., W. M., 10 years old. Normal birth and infancy. Illness began January 1946 with pain in the left side and fever to 103°. He was admitted to the hospital where physical and x-ray examinations indicated an effusion in the left pleural cavity. Aspiration of the left posterior chest yielded clear straw-colored liquid which was sterile on culture. During the ensuing six months there was improvement, but never complete recovery. Occasional attacks of pain with elevated temperature (101° to 102°) occurred. The x-ray shadow persisted (Fig. 1). He was readmitted to the hospital after one of these attacks on July 10, 1946. Examination—particularly physical (dullness, absence and fremitus; diminished breath sounds over the precordium and displacement of heart to the right) indicated either a tumor or encapsulated effusion in the left anterior chest. Other data, including laboratory findings, were irrelevant. An aspiration in the fourth anterior intercostal space left, was carried out and 120 cc. of liquid having the appearance of lime water and containing large numbers of particles of black and dark green soft, waxy material were withdrawn through a No. 15 gauge needle. Dermoid cyst was immediately suspected, but the examination of the material was inconclusive.

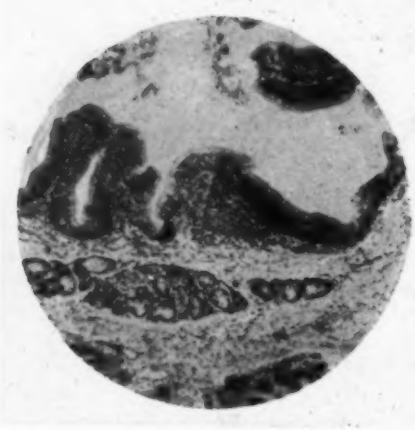
Operation: Exploration of the left pleural cavity was deemed advisable and was carried out on July 23. Through an antero-lateral approach in the fourth intercostal space, a tumor was found occupying the normal position of the heart. The incision was enlarged through this inter-

space and exposure improved by removing short segments of the fourth and fifth ribs posterior to the angle. The fifth rib was also sectioned anteriorly. The tumor was found to be densely adherent to the lower lobe, the diaphragm and the pericardium. It was freed by sharp and blunt dissection and found to arise by a two inch long pedicle from the anterolateral aspect of the mediastinum behind the pulmonary vessels, but in front of the bronchus. The pedicle was severed between clamps, permitting the removal of the tumor. The cyst wall was thin in some areas and a small part of its content was expressed by manipulation into the pleural cavity. This was washed out with sterile Ringer's solution. The lower lobe, which was adherent to the chest wall in the lower axillary area, was freed. A No. 22 F. mushroom catheter was inserted in the ninth posterior intercostal space for drainage. The chest wall was closed in layers. The lung was re-expanded by intratracheal pressure and aspiration of the air from the pleural cavity through the drainage catheter.

CASE IV, FIG. 1



CASE IV, FIG. 3



CASE IV, FIG. 2

Case IV, Fig. 1: H.N., W.M., age 10. Posteroanterior view of chest showing "residual effusion" in left base and displacement of heart to the right. (Previous x-ray, said to show obscurity of entire left hemithorax, not obtainable).

Case IV, Fig. 2: Section of tumor x460 showing adenomatous formations in a fibrous stroma together with columnar type epithelium suggesting bronchial of intestinal mucosa.

Case IV, Fig. 3: Posteroanterior view showing condition of chest, three months postoperative at which time patient was clinically well.

The drainage tube was removed on the third postoperative day and the chest was twice aspirated: on July 26, of 300 cc. of turbid amber fluid and on July 27, of 400 cc. of clear amber fluid. The patient was discharged on August 3, the eleventh postoperative day and has since remained well.

The pathological report is as follows: "The cyst measures 10x12 cm. in diameter, when partly emptied of fluid. The latter was cloudy with considerable yellowish-green powdery material showing no structure on smear, but evidently being keratin and debris; the fluid contained no pus and no hair. The cyst wall is 1x1.5 cm. in thickness, except where a polypoid structure consisting of fatty tissue, 7x8 cm. wide, is attached to it. The cyst wall around the base of this is fibrous, with a number of small cysts, containing mucoid material. No bone is found. The cyst lining is smooth, yellowish with a few short hairs, and a small amount of yellow sebaceous and fatty material.

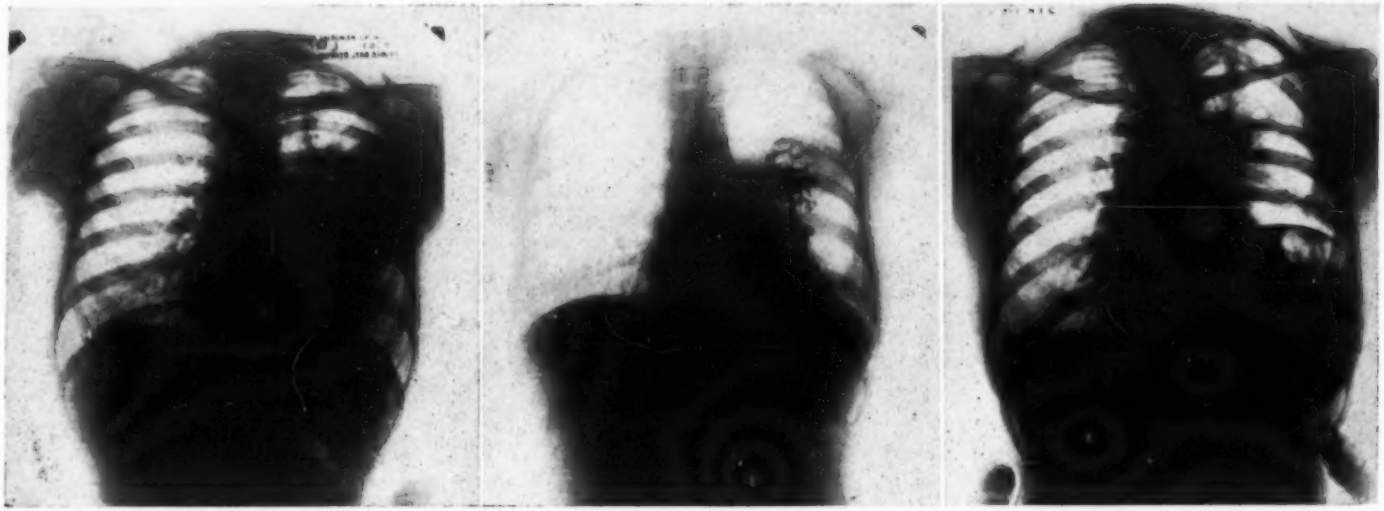
A number of areas are sectioned, and show a variety of structures. There is fibrous tissue, and smooth muscle, and adjacent groups of mucous glands—salivary gland or bronchial mucosa type—and areas of columnar respiratory type lining epithelium. A small area of cartilage is found; also small areas of intestinal type mucosa, considerable fat, and sebaceous glands. No areas of pancreatic or thyroid tissue are found. No areas of malignant epithelial hyperplasia are found (Fig. 2).

Diagnosis: Dermoid cyst, left pleural cavity."

The roentgenological condition of this patient's chest on October 18, 1946, is shown in Figure 3.

Case V: B.R., W.F., 34 years old. Normal birth and development. The patient was able to engage in the usual activities at school, although she noticed that her fingers were clubbed at the age of about 14 years. She had had a cough for "several years." In the summer of 1945, she went swimming in lower New York Bay. During one of these outings, she aspirated water into the lungs. On the following day, she had a chill, followed by a cough. She was admitted to the hospital on July 28, 1945, with a fever of 104°-105° and a cough productive of 8 ozs. of foul-smelling yellow sputum daily. R.B.C., 3,450,000; W.B.C., 19,400. There was evidence of consolidation of the left upper lobe and the x-ray film (Fig. 1) showed a large cavity and fluid level. Bronchoscopy was carried out on July 31, 1945, and drainage was found from the left upper lobe bronchus. Bacteriological and pathological study of aspirated material gave no important revelations. A diagnosis of lung abscess was made. Under conservative methods, the course of disease was unsatisfactory. Localizing films showed multiple fluid levels, which were not constant. A first stage pneumonostomy was performed on August 28, 1945. The pleura was free and a second stage was carried out on September 2, 1945. A large cavity was entered, but due to an exceptional amount of bleeding, de-roofing had to be limited to an opening of only about one-half inch in diameter.

Temperature returned to normal and cough almost disappeared. Although, after iodized oil injection (Fig. 2) it was felt that lobectomy would be necessary for complete cure, the patient was allowed to go home. Drainage continued from the sinus and finally, the patient exhibited a yellow hair about ¾ inch long, which she said she had coughed up. She was readmitted to the hospital on November 17, 1945. On November 20, 1945, in spite of considerable bleeding, the cavity was visually



CASE V, FIG. 1 CASE V, FIG. 2 CASE V, FIG. 3

Case V, Fig. 1: B.R., W.F., age 34. Admission x-ray showing large "abscess" cavity in left upper lobe with fluid level.—*Case V, Fig. 2:* X-ray of chest two months after drainage of "abscess" and following lipiodol injection into cavity through external opening. Note multiple loculations and communication with bronchi. The two masses of lipiodol below the safety pin are outside the body.—*Case V, Fig. 3:* Anteroposterior view of chest showing patient's condition six months after operation. At the time she was free of symptoms.

explored. It was found to be a part of a teratomatous cyst extending from the mediastinum into the left upper lobe, communicating with several bronchi and containing a large bundle of brownish-yellow hair. The sinus was allowed to close to a diameter of about ¼ inch and, on December 27, 1945, a left upper lobectomy, with removal of the mediastinal tumor and excision of the draining sinus, was carried out in a single stage operation.

Operative Technique: The thorax was opened along the 5th intercostal space. Individual ligation technique was used, but, due to the density of inflammatory reaction, was not completely feasible. In removing the mass from the mediastinum, the phrenic nerve was cut. The anterior sinus, after resection, was closed with muscle and skin and drainage was provided through the ninth posterior intercostal space.

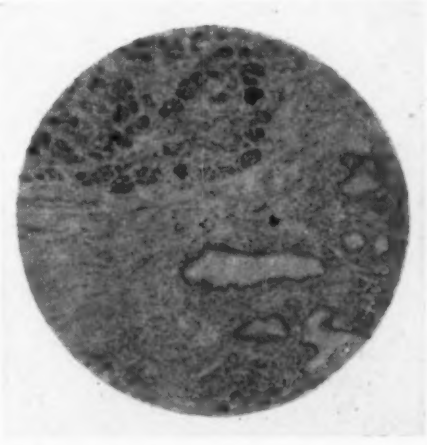
Convalescence was complicated by the development of an empyema, but was otherwise without important events. The patient was discharged on March 29, 1946 and has since remained well. The present appearance of her chest is shown in Figure 3.

Pathological examination is as follows (Fig. 4): "The specimen consists of the upper lobe, left lung, showing a well defined cyst 8x10 cm. wide, anteriorly, communicating with a bronchus. The lung tissue is reddish and firm. The cyst wall is firm and pale yellowish throughout and from 1.5-2 cm. in thickness. The inner surface is smooth. The cyst still contains a few strands of hair.

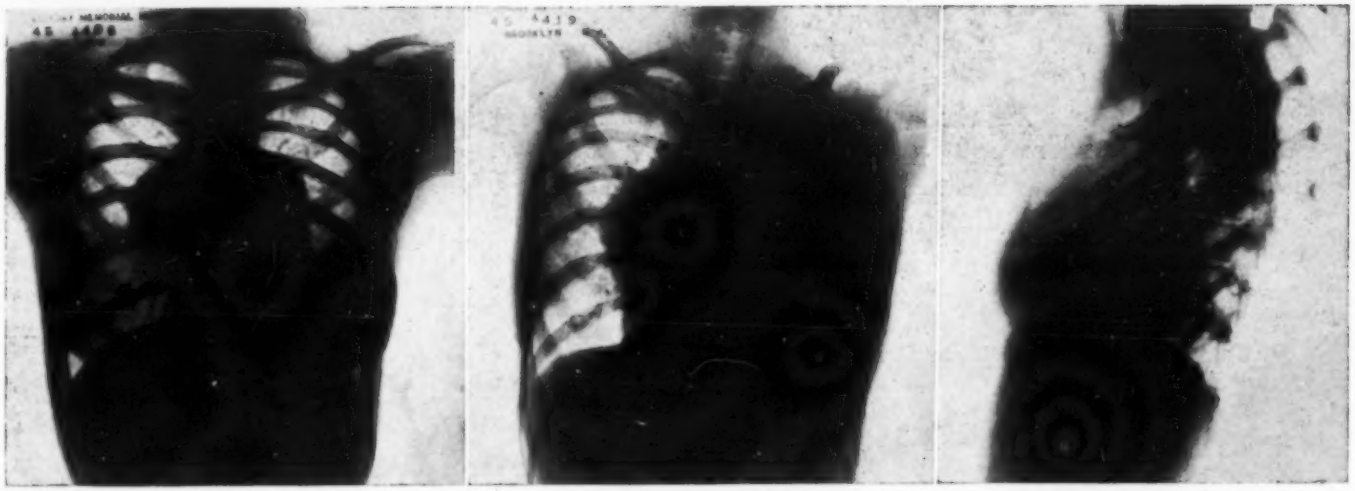
"Several areas are sectioned, and show fibrous tissue with squamous tissue, sebaceous gland tissue, and columnar mucus-secreting epithelium. In some parts, on the inner surface, the transition from columnar mucus-secreting epithelium to squamous epithelium is clearly defined. Other tissue rudiments, such as thyroid, are not found. No malignant change is found. Further sections of cyst wall show well defined pancreatic tissue, with islet tissue also.

Diagnosis: "Teratoma, left lung, upper lobe."

Case VI: E. S., W. F., age 16 years. Normal birth. No dyspnoea, no difficulty in infancy. She was able to engage in normal activities in school.



Case V, Fig. 4: Microscopic section x200 showing variety and irregularity of structural formation from glandular type to cystic arrangements lined by cells of differing morphology.

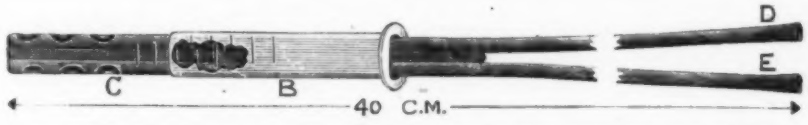


CASE VI, FIG. 1 CASE VI, FIG. 2 CASE VI, FIG. 3

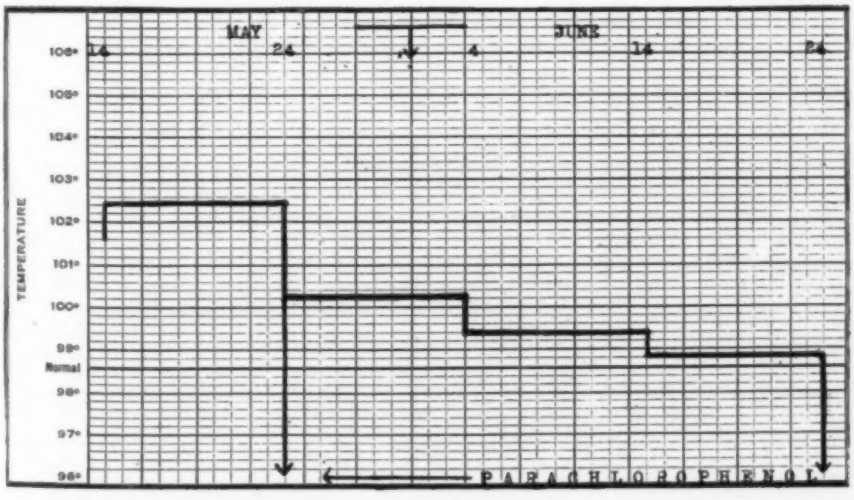
Case VI, Fig. 1: E.S., W.F., age 16. Posteroanterior view of chest showing a tumor occupying half the width of the thoracic cavity and beginning obscurity at the left base. This examination was made after deep x-ray therapy with no reduction in size of tumor.—*Case VI, Fig. 2:* Posteroanterior view of chest after a second episode of "pleurisy." Chest tap after this x-ray yielded 300 cc. of yellow turbid fluid sterile on culture.—*Case VI, Fig. 3:* Lateral view of chest following evacuation of fluid. Note double fluid levels, one at the third rib anterior and one at the ninth rib posterior.

On January 3, 1945, she suffered a severe pain in the left chest and was examined by a doctor who diagnosed it as "pneumonia." On January 12, an x-ray film was taken (Fig. 1). It showed a large mass in the mediastinal area extending into both lung fields. Electrocardiograph, blood Wassermann and other examinations were made without important revelations. Deep x-ray therapy was started on January 22, and the patient received a total dose of six treatments of 200 r. each (1200 r.), 92 K. V. and 10 M. A., following which her menses disappeared for a period of two years. The patient was admitted to the hospital on February 12, where a diagnosis of subacute bacterial endocarditis was made. This was followed by another episode of "pneumonia or pleurisy" (Fig. 2). She was admitted to a second hospital on April 9. On April 10, an aspiration was made of the left chest posteriorly, at which time 300 cc. of yellow, turbid fluid was obtained. The laboratory examination of this showed no growth. Intercostal drainage was instituted in the 8th left intercostal space posteriorly on April 19 and the tube was left in situ for two months without improvement. Her weight at this time was seventy-eight pounds.

The patient was examined on June 24, 1945, at which time the x-ray film showed a double fluid level (Fig. 3). There was a purulent drainage from the posterior thoracotomy wound. An exploratory thoracotomy was decided upon and this was carried out on July 5, at which time a large purulent collection was found in the posterior pleural cavity, with an anterior pocket lined by a thick fibrous membrane. No communica-



Case VI, Fig. 4: Two way catheter used in this case for the maintenance of constant irrigation of the pleural cavity under constant negative pressure.

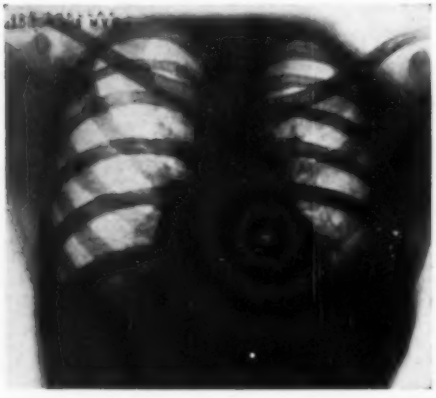


Case VI, Fig. 5: Postoperative temperature record showing average elevation in the day periods beginning ten days before and ending thirty days after the use of parachlorophenol 1:1000 to mediastinal pyocyaneus infected wound.

tion with the mediastinum could be found. The wall of the anterior pocket was divided by sharp dissection. In view of the chronicity of the condition, a large drainage opening was left.

Her condition still failed to show improvement and, about the middle of July, edema of the lower extremities developed. This edema gradually spread upward until the whole body was involved. At the same time, frequent bouts of fever from 103°-104° occurred, accompanied by large quantities of blood in the urine. Urinalysis at this time showed numerous R. B. Cells, granular casts and a one plus albumin. Serum proteins: albumin, 3.7 per cent and globulin, 3.2 per cent. Congo red test was negative. A diagnosis of acute nephritis was made.

On August 8, treatment of the empyema was begun with the author's two-way catheter (Fig. 4) in the wound and constant saline irrigation was employed under negative pressure. Gradual improvement was noted. On September 16, the patient was discharged without edema, but was not considered a good surgical risk at the time. The posterior thoracic cavity was obliterated, but a small sinus from the anterior pocket still remained. Throughout the course of this admission, there was a persistent pyocyaneus infection, both in the wound and on the dressing. After seven weeks' rest at home, the patient was readmitted with a chest wall abscess below the left breast. On November 14, this abscess was drained by resection of the fourth and fifth ribs, both of which were found to be denuded of periosteum. The sinus was followed to the level of the second interspace and through and through drainage was established by Penrose tube. Pure cultures of pyocyaneus were obtained from the wound. All the usual agents were employed to combat this infection without result. The Penrose tube was removed on November 26 and the wound packed with a solution of streptomycin (10,000 units per gram) with an immediate clearing of the typical color and odor of this organism from the dressing. The lower wound closed rapidly. The upper remained open and continued to yield pyocyaneus on culture. From this time onward,



CASE VI, FIG. 6

CASE VI, FIG. 7

Case VI, Fig. 6: Posteroanterior projection of chest two months after discharge from hospital. The wound remained completely healed and the patient was symptom free.—*Case VI, Fig. 7:* Microscopic section of tumor demonstrating glandular formation with nearby hollow structure lined by bronchial type epithelium with underlying lymphoid tissue.

the growth did not appear to be influenced by streptomycin, although the wound was packed with paste containing a concentration of 100,000 units per cc. of the agent. On January 28, 1946, the residual sinus was explored after injection of methylene blue and was found to enter the mediastinum. An exposure was obtained by resection of segments of the second and third ribs and a nodule was removed for microscopic examination. This showed a mixture of tissues of entodermal, ectodermal and mesodermal origin—characteristic of teratoma. Again, the ineffectiveness of streptomycin was demonstrated, indicating the rapidity and permanency with which pyocyaneus developed resistance to this preparation. There was, however, further improvement in the patient's condition. A total gain in weight of twenty-four pounds was registered. Temperature was normal. The urine was clear. The patient was discharged on March 28 with a drainage tube in the anterior mediastinum and a persistent pyocyaneus infection. On May 6, she was readmitted for removal of the tumor. Her general condition was now excellent: weight, 112 lbs., no edema, urine clear, and R. B. C. count 4,000,000. The operation was carried out on May 14, under intratracheal gas-oxygen and ether. A "U" shaped incision was made, based on the left border of the sternum. The gladiolus sterni was turned to the right by fracturing the cartilages on that side. In effecting this exposure, it was necessary to remove the periosteum from the under surface of the bone, since this was the only line of cleavage that could be safely followed. The tumor, which was the size of a small, somewhat flattened orange and in the shape of an hour-glass, was found to be bound down by dense adhesions on all sides—to the mediastinal structures, the pericardium and the great vessels. The upper half was below the manubrium sterni and seemed to be incorporated with the thymus. Both segments required a tedious process of both sharp and blunt dissection, during which the mediastinal pleura on the right was opened. There was momentary dyspnoea and cyanosis, which was rapidly overcome by management of pressure in the anaesthesia circuit. An attempt to repair the opening proved futile, due to the fragility and looseness of the structures available. The wound was packed with loose gauze containing 100,000 units of penicillin over a Penrose drain. The gladiolus sterni was brought back into position and stabilized satisfactorily by skin sutures only. A No. 24 F. mushroom catheter was inserted in the ninth posterior intercostal space for drainage of the right pleural cavity.

The postoperative course was as follows: The temperature rose to 103° and there remained for the first ten postoperative days (Fig. 5). There was a predominating growth of pyocyaneus from the wound and gradual sloughing of mediastinal fat, together with spread of the infection, although the opening into the right mediastinal pleura closed during this time. A large number of chemical agents were used for packing and irrigation—including streptomycin paste up to 100,000 units per cc.—but without effect. At this point, it was possible to obtain a supply of parachlorophenol for the purpose. One application of solution 1:2000 was used to test the reaction to the skin and tissues. There was none. The dressing was then changed three times daily, using a dilution of 1:1000 of the agent. There was immediate improvement. Within forty-eight hours, sloughing had stopped and healthy granulations began to appear. The pleural cavity was also irrigated through the tube with the same solution. Despite rapid and continued improvement, pyocyaneus was cultured from both the mediastinal wound and the pleural cavity

for the next three weeks (June 14). On June 15, the tube was removed from the pleural cavity. The sinus was kept open by a grooved director for a few days. The anterior dressing was changed once daily. The patient was discharged on June 27 and has since remained well. The appearance of the chest on August 28 is shown in Figure 6.

Pathological Report: "Gross Examination: The specimen is 9 cm. long, 5 cm. wide, smooth posteriorly and presents a number of glistening, smooth nodules on the anterior surface. The nodules vary in size, measuring 1.5x2.5x3.0 cm. in diameter, with wide bases. Several nodules are pale throughout and suggest fatty and fibrous tissue. The surface shows a few hairs. Other nodules are reddish on section. Bony areas are not encountered. There is also a second specimen, 3.5x3.2 cm. in size, reddish on section.

"Microscopic examination: A number of areas are sectioned (Fig. 7) and these show the varied histological structure which may be found in teratomata. The pale nodules show a squamous surface layer, then fibrous tissue and fat, and a few acini of columnar intestinal type epithelium. Small areas of cartilage are found. Other parts show considerable glandular tissue suggestive of fetal thyroid or of pancreas, as noted in the biopsy specimen. Adjacent to the glandular tissue is a small lumen lined at one side with columnar bronchial type epithelium, then changing to squamous epithelium with subepithelial lymphoid tissue. The tissue examined shows no evidence of malignancy.

"The second specimen—reddish—shows thymic tissue—lymphoid tissue with scattered Hassall's corpuscles. There is also some fibrous tissue here, but teratomatous elements are not found. Inflammatory changes are not found, except for slight lymphocytic infiltration in an area on the surface of a nodule, where the squamous epithelium appears eroded.

"Diagnosis: Teratoma of mediastinum, benign. Thymus gland."

SUMMARY

1) Six cases of tumor of the anterior mediastinum are reported. Five of these were treated surgically with clinical cures in all cases.

2) One of them was an "undifferentiated tumor" in a new born infant producing obstructive dyspnoea and death. This patient also could probably have been saved if permission for operation had been granted.

3) The importance of avoiding complications particularly infections by early and correct diagnosis is demonstrated.

4) X-ray examination is shown to be the only method by which early diagnosis can be made. It is urged that the same importance be assigned to nontuberculous as to tuberculous diseases of the chest in the application of the technique of mass x-ray survey and follow-up.

5) The value and necessity of measures designed to save life or prepare the patient for curative surgery are brought out. These may include the preliminary drainage of the cystic portions of teratomata or dermoids.

6) Streptomycin is shown to be an effective agent for the control of pyocyaneus infection in its pathogenic phase if there are

no sinuses or pockets and if the entire infected area can be reached by the first applications. The organism, however, develops a rapid resistance to the agent if it is not immediately destroyed.

7) The value of parachlorophenol in the control of pyocyaneus infection is demonstrated. This agent appears to exert a bacteriostatic action upon the organism. There is no reaction from the body tissues and the effectiveness of the drug does not appear to be diminished by continued use.

Pathological report on Case II was prepared by Dr. M. Freund, on all other cases by Dr. M. Penke.

Anaesthesia in Case II was administered by Dr. Joseph Takach, in all other cases by Dr. M. Swick.

I am indebted to Dr. F. Meleney for assistance in obtaining parachlorophenol and for his advice in respect to its use as well as to Merck and Co. who contributed a generous supply of the drug.

I wish, also, to pay a debt of gratitude to the following physicians and surgeons for advice and assistance freely given: Dr. Thomas Galvin, Dr. William Watson, Dr. Edward Robitzek and Dr. Kenneth Kahn.

RESUMEN

1) Se informa sobre seis casos de tumores del mediastino anterior. Se trató quirúrgicamente a cinco con curaciones clínicas en todos los casos.

2) Uno de ellos fue un "tumor no diferenciado" en un recién nacido, que produjo disnea obstructiva y muerte. Probablemente se podría haber salvado también a este paciente si se hubiera otorgado permiso para la operación.

3) Se demuestra la importancia de evitar complicaciones, particularmente infecciones, mediante el diagnóstico temprano y correcto.

4) Se demuestra que el examen radiográfico es el único método por medio del cual se puede hacer el diagnóstico temprano. Se urge que se le dé la misma importancia a las enfermedades del pecho no tuberculosas como a las tuberculosas en la aplicación de la técnica de censos radiográficos colectivos y observaciones consecutivas.

5) Se ponen de manifiesto el valor y la necesidad de emplear medidas de salvar la vida y preparar al paciente para la cirugía curativa. Estas medidas pueden incluir la canalización preliminar de las partes quísticas de teratomas y dermoides.

6) Se demuestra que la estreptomicina es un agente eficaz en el control de infección piociánica en su fase patogénica, si no existen fístulas o bolsillos y si las primeras aplicaciones pueden alcanzar a la entera zona infectada. Sin embargo, el gérmen desarrolla una rápida resistencia al agente si no se le destruye inmediatamente.

7) Se demuestra el valor del paraclorofenol en el control de infección piociánica. Este agente parece ajercer una acción bacteriostática sobre el gérmen. No ocurre reacción en los tejidos del cuerpo y la droga no parece perder su eficacia con el uso continuado.

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The Combined Thoracoplasty*

GUSTAV MAURER, M.D., F.C.C.P.
Schatzalp Sanatorium, Davos, Switzerland

We shall begin our address by analysing the *weak points of thoracoplasty*. As a general rule, we must expect some after-effects of thoracoplasty which include cardiac disturbances, retention of bronchial secretions, reactivation of the lesion and fresh disseminations. The surgical shock is first held responsible for these complications, and perhaps, to a greater degree the sudden pulmonary collapse, which results not only in an abrupt tuberculin shock, but sometimes in the release of a veritable flood of toxins. I am far from wishing to contradict these conclusions drawn from surgical risks, yet, I see the principal danger in the *unphysiological state of the thorax, caused by thoracoplasty*. Every surgical intervention which alters the anatomy of a certain organ, harms it and disturbs its functions.

Thoracoplasty creates difficulty in breathing, this being of a paradoxical nature in the resected part. The mediastinum having lost its normal balance between the two lungs, now follows the paradoxical movements of the operated side, and thus the breathing capacity even of the sound lung is reduced. The inevitable paradoxical retractions and expansions of the unsupported thoracic wall set up a *traction irritation upon cortical lesions*. This mechanical trauma reaches its height in the region between the rib stumps and the resected part. Because an active and superficial focus is partially attached to the rib stump and the remainder is situated in the resected region, with each breath and particularly with each cough, half the lesion is retracted while the other half is inflated.

An even greater danger for the patient is the physiopathologic disturbance which results from the *difficulties in expectoration*. Let us consider the normal cough. The glottis is closed, the diaphragm descends, abdominal pressure at its height and then the ribs move inward abruptly. By this means, a reduction of the thoracic space takes place, causing maximal tension in the bronchial system. Now by the sudden opening of the glottis, air and sputum under pressure are driven out, one might almost say spouted up through the trachea. The question now arises of how

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does this cough and expectoration materialize after thoracoplasty. The increase in intratracheal pressure, indispensable to coughing, is hampered because the thoracic cage, in the resected area, cannot contract and worse still, is blown out like a hernia. We see therefore that this lack of pressure can only be compensated for, by strengthening the coughing process, so that free expectoration is nevertheless ensured.

Discomfort after the operation is not only distressing, but also constitutes a considerable danger. About the first fortnight after thoracoplasty coughing hurts the patient because the anterior rib stumps dig into the wound. He inevitably restrains himself so as to avoid this pain. The patient who is bound to have a faulty mechanical action of expectoration, should, on the contrary, attempt to intensify the coughing. *Retention of sputum,* is therefore to be expected, and it will result in toxemia, which in turn is further aggravated by mechanical irritation and reactivity of superficial lesions. Increased tissue destruction, fresh disseminations, specific bronchial pneumonia are all to be noticed. It is truly a vicious circle, which persists until the ribs are regenerated. *Thoracoplasty is, in my opinion, an unphysiological intervention, and it is to this fact that I especially attribute postoperative pathologic changes.*

From this conception, I have looked for a collapse therapy method which will safeguard to the utmost limit the physiologic status and in consequence, pays due regard to the thoracic and pulmonary functions. In theory, a natural and adequate covering is obtained in the region of the scapula, and there only. In practice, this smali thoracoplasty with the posterior-lateral part of four or five upper ribs only, does not get us very far, as the resulting pulmonary collapse is not in itself of sufficient effectiveness. We must therefore endeavour to complete it by other means. As accessory collapse-therapeutic factors I suggest:

1. *The resection of five to six upper intercostal nerves (with the exception of the first one).* We thus obtain a paralysis of the frontal costal rib stumps, which now drop and turn inwards in conjunction with the retraction of the lung. We get therefore, a decrease of the thoracic cage in its anterior-lateral section and, a matter of no little importance, an insensibility of the whole field of operation.

2. *The luxation of the shoulder blade into the thorax.* The scapula can well provide the support needed after the removal of the four to five upper ribs, and in addition, gives us an osteo-muscular filling by diminishing in an effective manner the posterior-lateral region of the thoracic cage down to the eighth rib.

3. To facilitate the enclosure of the shoulder blade, we must

create a pneumolysis, starting from the first intact rib, in other words, from the fifth or sixth and continuing towards the base. *This extrapleural basal detachment* can be enlarged according to the general position and dimension of lower lesions.

The characteristics of the combined thoracoplasty are as follows: a considerable diminution of the thoracic cage in its upper part, with complete cohesion of every particle so there are no paradoxical movements. Breathing and circulation are in no way hindered, the patient may cough painlessly and expectorate freely. We have no reason to fear aggravation of the lesion, fresh disseminations or bronchial pneumonia. *None of the four collapse-therapeutic factors of this method meets our requirements of its individual merits, but by uniting them, we gain an ideal relaxation of the lung and safeguard the physiologic state of this organ.*

Combined thoracoplasty should be done in two stages. For grave cases it is better to divide the shock. The first stage (i.e., the resection of the third and fourth ribs as well as their intercostal nerves), is a test not only of the reactivity of the lesions in question, but of the effect upon a process in the other lung and upon extrapulmonary foci. *Besides these, there is an absolutely primordial reason which obliges us to operate in two stages.* After the first stage, the pleura in the resected part thickens and becomes as hard as cardboard. The vasculo-muscular network and the wound exsudate cause the thickening of the pleura. At the second stage, the collapsed lung protected by the hardened surface slips lower down. We thus avoid the dangerous trauma which the movements of the intrathoracic scapula could cause to the cortical lesions.

One mechanical impediment of thoracoplasty is the space between the *scapula, mediastinum and clavicle.* During the resorption and organization of the wound exudate, the collapsed lung is irresistably drawn towards this noxious space which has the form of a trihedron, the *dead trihedron of thoracoplasty* (a partial reexpansion of the lung apex after the intervention is unavoidable since nature abhors a vacuum). The inconveniences arising from the empty space between the shoulder blade and the resected thoracic dome are well known. Archibald uses a muscular filling; Lilienthal, fat; DeWinter suggested an intrathoracic luxation of the shoulder blade, but had to give it up because in some cases severe pain immobilized the scapula and in others the cortical lesions were traumatized; Adams, Steinke, John Alexander, Villani, Coryllos, Holman, Torning, Overholt, advise the resection of the lower part of the scapula to avoid these drawbacks. The intrathoracic dislocation of the scapula allows us to diminish the "dead trihedron." The shoulder blade, by drawing nearer to the

clavicle, closes this vacuum from behind like a door; the acromio-clavicular joint acting as a hinge.

To sum up, the intrathoracic scapula plays two parts. It serves both as a filling and as a support for the resected chest-wall. The resection of the intercostal nerves has a triple function. First the wound is made insensible, a great benefit to the patient. By this means, expectoration is eased. Furthermore, intercostal neuralgia arising from the pressure or the pendulous action of the intrathoracic shoulder blade is thus avoided. With the resection of the intercostal nerves, we therefore prevent the fixation and the consequent stiffening of the arm through pain. Lastly, the nerve resection is a therapeutic factor, par excellence, since the costal breathing of the hemithorax is reduced. Basal pneumolysis completes the collapse in the lower parts. This then is the description of the weak points of thoracoplasty and of their remedy. *But it is only by extending the field of indication, by improving the results and by reducing the complications,* that we can prove the superiority of our method. Otherwise all analysis is and remains purely theoretical.

We are often receiving patients for treatment who had been refused active intervention. We had in 120 combined plastic operations, only 33 cases which had the usual indication and 87 cases where the indication was a risky one. The results from these 120 patients are as follows:

Good, 70 per cent: Inactivity of all tuberculous lesions, intra or extrapulmonary, negative for tubercle bacilli.

Incomplete, 16.7 per cent: Healing perhaps still possible by further surgical treatment.

Direct deaths, 3.3 per cent: Operation or too risky indication responsible; patient dying within six months after the intervention.

Indirect deaths, 5.8 per cent: Without any relation to the intervention, but aggravation of tuberculosis could not be stopped despite thoracoplasty; death from six months to years after operation.

Independent deaths, 4.2 per cent: War, accidents, suicides, etc.

We are not considering the surgical technique, because in 1946 it has already been dealt with by Dr. Willenegger, formerly a surgeon on our staff, in Helvetica Chirurgica Acta, but we should like to draw your attention to several advantages of the combined thoracoplasty.

We get very few uni- or contralateral disseminations. We have even been able to perform thoracoplasty upon cases with a cavity in the opposite lung. We hardly ever notice an aggravation of extrapulmonary foci after the operation.

Steinlin proved by spirography, that as a rule *combined thora-*

coplasty in no way reduces the respiratory function, and there is no lack of oxygen, a special advantage indeed for double collapse therapy. On the contrary, Cournand and Dickinson emphasize the fact that in every case with diminished pulmonary function before thoracoplasty, this function is afterwards still further reduced.

The arm movements are as easy as after other thoracoplastic methods: The absence of severe surgical shock is striking. Sixty-six operations were performed upon out-patients, all of whom were able to leave the Sanatorium on foot, on an average of 7.4 days after the operation.

The fact that out of 268 operations, we have not had a single case of infection is equally striking, although our asepsis is nothing unusual. The healing per primam must be attributed to the surgical method. The intrathoracic shoulder blade acts as a muscle filling, in accordance with the surgical treatment of accidents, where to prevent infection, a gaping wound is protected as far as possible by a muscle transplantation.

The method is at least as effective as an eight rib thoracoplasty. Thanks to the three accessory collapse-therapeutic factors, lesions below and anterior to the resected ribs can still heal. Should a cavity remain open, combined thoracoplasty can be easily enlarged, as it constitutes a key position in collapse mechanism, and there is nothing to prevent us from continuing with the vertical and horizontal reduction of the thorax.

Some Special Therapeutic Problems

Complete basal pneumolysis to avoid postoperative reactivation of basal foci; example:

A small cavity was present in the right apex, and there was slight dissemination in the base of the same lung. The x-ray film, taken 9 days after combined thoracoplasty, showed a complete basal pneumolysis. The picture, taken 9 months after the operation revealed the resorption of the pneumolysis exudate, the regression of the basal pulmonary dissemination, the apical cavity closed, and a matter of no little importance, the respiratory function completely regained.

Basal pneumolysis gives a temporary collapse of 4 to 6 weeks only, most useful indeed *to prevent postoperative aggravation of an affected lung base.* If therapeutically insufficient, this temporary pneumolysis can be completed or replaced by phrenic nerve crush plus pneumoperitoneum.

Combined thoracoplasty and tuberculous pneumonia; example:

Evidence of caseous pneumonia of the left lung was seen on the x-ray picture and a special film showed an extreme destruction of this caseous lung. After preparation of phrenic nerve crush plus pneumoperitoneum, ten months later, thoracoplasty was performed. The efficacy of the basal pneumolysis would have been too temporary and therefore insufficient for this severe lung process. The x-ray picture after the intervention

showed a remarkable collapse of the operated side. There was no further sign of active tuberculosis and the patient has been working for the last two years.

Tuberculous pneumonia does not necessarily exclude our operation.

Combined thoracoplasty and the fatal tuberculous triad: lung, larynx and abdomen; example:

This patient had specific ulcerated larynx, specific peritonitis, and caseous pneumonia in the right upper lobe (unsuccessfully treated by phrenicotomy) and also a small cavity below the left clavicle. Tomography of the right apex showed various cavities in the caseous upper lobe and the tomogram of the left lung revealed the said small cavity. The picture, 9 months after plastic operation, showed a perfect left lung and an ideal collapse of the right one. The larynx was healed and there were no further abdominal symptoms. The patient gained nearly 28 lbs.

Tuberculosis of the lung, larynx and abdomen is not always fatal.

Combined thoracoplasty and double collapse therapy; example:

The x-ray film showed a right apicolysis with wax filling for a subclavicular small cavity, and a big cavity with fluid level in the left lung, as well as basal disseminations, and aftermaths of pleurisy on both diaphragms. (A very bad case indeed, as we only use these wax fillings when there is no other alternative). Eleven weeks after the right apicolysis, thoracoplasty was performed on the left side. The x-ray picture shows a remarkable collapse in the upper part of the left lung and resorption of the basal disseminations, in short, inactivity of the tuberculous process.

Combined thoracoplasty as a rule does not reduce the breathing. It gives an individual aimed collapse, strictly limited to the lesions, which are nevertheless out of function. For instance, an artificial pneumothorax in the opposite side can be well supported even when an exudate arises, there is still sufficient respiratory function in the base of the plastic lung.

SUMMARY

A) *Weak Points of Thoracoplasty:* Breathing capacity reduced by paradoxical movements of operated side, paradoxical retractions and expansion of the unsupported thoracic wall, consequently traction irritation of cortical lesions. Difficulties in expectoration, (reasons thereof intensified by painful coughing). Consequences: retention of sputum, increase of lesions and danger of further pulmonary and extrapulmonary spreads.

B) *Prevention of these Weak Points:* Restriction of thoracoplasty to the posterior-lateral parts of the four to five upper ribs, the region which is covered by the shoulder blade. In this region paradoxical movements are thus avoided, but therapeutic effect is insufficient, therefore accessory factors of collapse therapy are necessary.

C) *The Combined Collapse Method:*

1) Resection of the posterior-lateral parts of the four to five upper ribs and painting of the periosteum with a 10 per cent formalin solution (to avoid rib regeneration and to make retraction of lesions possible even later on).

2) Resection of the five to six upper intercostal nerves with the exception of the first (avoiding pain of and breathing with the anterior chest wall).

3) Pneumolysis from the first intact rib, in other words, from the fifth or sixth towards the base (sufficient in size for the luxation of the shoulder blade and to be enlarged according to the position and dimensions of the lesion).

4) Intrathoracic luxation of the shoulder blade as an osteomuscular filling and as an effective support for the resected thoacic region.

D) *Chief Characteristics of Combined Thoracoplasty:* No paradoxical breathing, normal respiratory functions unrestricted, no pain, no retention of sputum, no irritations of lesions, no further tuberculous spreads, surgical shock reduced to minimum, number of cases hitherto denied active treatment, operated, arm action almost unchanged, total absence of infection, results justify method, equal advantage with eight rib thoracoplasty, key position in collapse mechanism, no obstacle for further horizontal or vertical extension of thoracoplasty.

RESUMEN

A) *Defectos de la Toracoplastia:* Disminución de la capacidad respiratoria por los movimientos paradójicos del lado operado, contracción y expansión paradójicas de la pared torácica sin apoyo y, por consiguiente, irritación por tracción de lesiones corticales. Dificultad de expectorar (agravada por el dolor que da la tos). Resultados: retención de esputo, aumento de las lesiones y peligro de más propagaciones pulmonares y extrapulmonares.

B) *Prevención de estos Defectos:* Restricción de la toracoplastia a las partes postero-laterales de las cuatro o cinco costillas superiores, la región que está cubierta por el omoplato. Se evitan así los movimientos paradójicos de esta región, pero es insuficiente el efecto terapéutico y, por consiguiente, se necesitan factores accesorios de colapsoterapia.

C) *El Método de Colapso Combinado:*

1) Resección de las partes postero-laterales de las cuatro o cinco costillas superiores y aplicación al periostio de una solución de formalina al 10 por ciento (para evitar la regeneración de las costillas y para que sea posible la retracción de las lesiones, aún más tarde).

2) Resección de los cinco o seis nervios intercostales superiores con excepción del primero (lo que evita el dolor y los movimientos respiratorios de la pared torácica anterior).

3) Neumonolisis desde la primera costilla intacta, es decir, de la quinta o sexta hacia la base (de suficiente tamaño para la luxación del omoplato y agrandada de acuerdo con la ubicación y dimensiones de la lesión).

4) Luxación intratorácica del omoplato como un relleno osteomuscular y para el apoyo efectivo de la región torácica resecada.

D) *Características Principales de la Toracoplastia Combinada:* Ausencia de la respiración paradójica; ninguna restricción de las funciones respiratorias normales; ningún dolor, ni retención de esputo, ni irritación de las lesiones; no más propagaciones tuberculosas; reducción al mínimo del choque quirúrgico; el número de casos operados, a los que hasta ahora se les había negado el tratamiento activo; casi ninguna alteración de los movimientos del brazo; completa ausencia de infección; los resultados justifican el método; tiene las mismas ventajas que una toracoplastia de ocho costillas; la posición de clave en el mecanismo de colapso; ningún obstáculo a la subsiguiente extensión horizontal o vertical de la toracoplastia.

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Discussion

PAPKEN S. MUGRDITCHIAN, M.D., F.C.C.P.
Beirut, Lebanon

I had the privilege of following up Dr. Maurer's first operation by this new method, done in 1940 on one of my patients.

The operation described today has evolved from the fundamental idea of making a limited 4 to 5 rib thoracoplasty with bedding in of the scapula, with the dual object of achieving an optimal collapse and at the same time preserving good lung tissue. Then, the 1st stage was started from the lowermost ribs and a limited extrapleural pocket established in which the lower angle of the scapula was inserted in front of the remaining ribs.

Today Dr. Maurer has sensibly changed his operative technic and widened the scope of indications to a great number of cases, suitable for present standard thoracoplasty procedures. He now performs an intercostal neurectomy on nerves corresponding to the resected ribs, with the exception of the 1st nerve, thus immobilizing the upper chest wall and encouraging trophic changes in the remaining long anterior rib stumps whose periostei are shelled off and painted with 10 per cent formalin. At the same time an extrapleural pocket is established below the decostalized chest parieties to degrees varying with the extent and location of

the pulmonary lesions. This in turn tends to cause a concentric relaxation of the lung during the postoperative period by the collection of serum and air in the pocket that is allowed to absorb spontaneously.

The avoidance of extensive anterior chest wall decostalization and diminution of pain by neurectomy will contribute sensibly towards a less stormy and less complicating postoperative course. But, I do not think this operation can replace the standard thoracoplasty for the average case.

In large upper cavities and extensive old disease in the lower lobe, this operation will often be inadequate even with simultaneous phrenic nerve paralysis. One should not overlook also the lowering of ventilatory function subsequent to the immobilization of the upper chest wall. One of my cases with multiple intercostal neurectomy died from a contralateral spontaneous pneumothorax before help could heach him. The operation will find its main application, I believe, in the borderline bilateral subacute cases with lesions extending noticeably towards the base, this being a more effective collapse than the intercostal neurectomy alone. For limited upper lesions and especially in the presence of bilateral disease I have done a variant of the same operation on 15 patients since 1941, representing about 8 per cent of all thoracoplasties done. Longer pieces of upper ribs are removed, with an extrafacial apicolysis if indicated. Usually the 5 upper ribs are resected and neurectomy done on the last decostalized intercostal zone and the one immediately below it. The bedding in of the lower angle of the scapula is done in preference to scapulectomy, as it might contribute to the collapse of the frequently affected lower lobe apex; while the accompanying extrapleural pocket is accommodated in extent, usually limited, to the minor lesions tending to extend downward. When these lesions are more important, it is preferred to resect more ribs. Most of these 15 patients had major bilateral collapse operations. In all cases, the cavities were mechanically closed.

12 apparently cured and arrested.
2 quiescent.
1 died from massive contralateral hemorrhage 3 years subsequently.

Aesthetic results were good: no deformity, one questionable scoliosis; 2 cases complain of little pain. Arm function is good in all patients.

This variation of thoracoplasty developed by Dr. Maurer is a noteworthy addition to our armamentarium of collapse therapy methods. It will find its application in a limited number of properly chosen cases.

Cardiospasm with Associated Pulmonary Disease

T. G. HEATON, M.D., F.C.C.P.
Toronto, Canada

A recent report by Rothstein and Pirkle[1] calls attention to cardiospasm as a condition predisposing to the aspiration of foreign material into the lungs. They report a case of subacute pulmonary disease in a woman with cardiospasm and refer to a few other reports in the literature. Their patient had acid fast bacilli found on culture of sputum and these were considered not to be tubercle bacilli. They quote three cases from the literature in all of which acid fast bacilli other than the tubercle bacillus had been cultured from the sputum in the presence of subacute or chronic disease of the lung associated with cardiospasm.

On the basis of two cases from the literature and their own case they suggest the syndrome may be radiologically recognizable ("Bilateral upper lobe dense infiltrations with highlights, associated with widening to the right of the mediastinal shadow. The pulmonary lesion is found chiefly at the bases of the upper lobes and so reaches down farther on the left. Coarse infiltration may be present in the lower lung fields").

CASE REPORT

Mrs. C., age 51 in 1946. This woman first developed symptoms of cardiospasm in 1919 and had very severe symptoms, which have continued with diminishing frequency and severity. She had pneumonia first in 1918, with influenza. In 1933, she suffered a severe illness diagnosed, without x-ray, as pneumonia with lung abscess and in 1937, had an acute pneumonic illness which failed to resolve. She was investigated in a sanatorium in 1938 and was told she was tuberculous and advised to return to the sanatorium. As a result of this advice she consulted the writer who gave her his opinion that the disease was a nontuberculous pulmonary fibrosis, but without recognition at that time of its relation to cardiospasm.

At the present time, she has little cough and scanty sputum and the radiological appearances are illustrated. Acid fast bacilli have not been found. In the last five years, rheumatoid arthritis has developed and led to permanent invalidism.

This case is presented as a further example of fibrosing pneumonitis associated with cardiospasm and thought related to the aspiration of foreign material overflowing into the trachea from the oesophagus as a result of the cardiospasm.

SUMMARY

A case is reported in which cardiospasm is associated with chronic pulmonary disease and in which the chest x-ray has a characteristic appearance. The literature contains reports of similar cases. The pulmonary disease is thought to be caused by aspiration of contents from the dilated oesophagus.

RESUMEN

Se informa sobre un caso de cardioespasmo asociado con enfermedad pulmonar crónica en el que la radiografía del tórax reveló un aspecto característico. Se encuentran informes semejantes en la literatura. Se cree que la aspiración del contenido del esófago ensanchado causa la enfermedad pulmonar.

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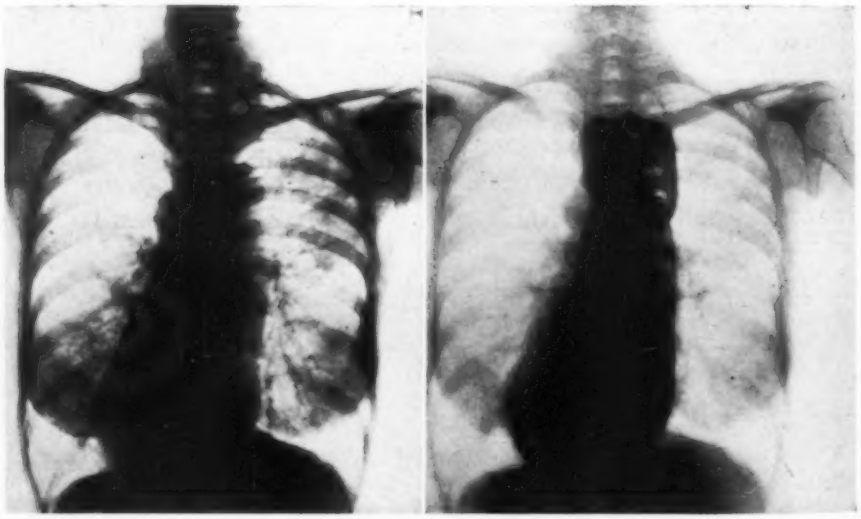

FIGURE 1 FIGURE 2

FIGURE 1: Showing the chronic pulmonary infiltration, and the line indicating the position of the oesophagus curving downwards and inwards from the left apex.—FIGURE 2: Showing the barium-filled oesophagus.

Bronchial Adenoma

WILLIAM E. HOWES, M.D.
Brooklyn, New York

Benign bronchial adenoma was originally identified from necropsy material by Mueller[1] in 1882. Before the advent of bronchoscopy such a tumor was only recognized, if at all, at the autopsy table. Even today these tumors are frequently mistaken by clinicians for either carcinoma or pulmonary tuberculosis.

Peterson[2] in 1936 was able to collect but 43 cases from the literature. Bronchial adenomata are not however as uncommon as these figures might indicate. Recheck of the literature through 1946, a total of 278 cases can be positively identified. Interestingly 235 of these cases have been reported in the last decade, i.e., since 1936. Relatively large series have been reported by Foster-Carter[3] 22, Clerk and Bucher[4] 35, Jackson and Konzelman[5] 20, and Moersch and Tinney[6] 38.

Of all benign intrabronchial tumors, the adenoma is by far the most common. Other benign intraluminal growths which may occur include: polyp, fibroma, chondroma, lipoma, papilloma, lymphoma, thyroid adenoma and mixed tumor.

History: In most instances the patient tells of repeated attacks of cough associated with chills and fever.

Symptoms: Hemoptysis is the most startling symptom. Hemorrhage is apt to occur suddenly during a coughing seizure. Bleeding is dramatic in its suddenness and severity, though it usually ceases as suddenly as it appears. Cough is apt to be persistent and unproductive. Temperature is often secondary to bronchitis or pneumonitis due to temporary or permanent blockage.

Physical findings: Physical findings depend on the proportionate air blockage by the tumor in the lumen of the affected bronchus (Fig. 1). Three distinct stages may be classified:

Stage I: The tumefaction is small and does not obstruct the airway. Physical findings are entirely negative at this time.

Stage II: The tumor all but blocks the bronchial lumen. Air is sucked past the growth and is trapped distally due to the fact that the inspiratory stroke of the diaphragm is more powerful than the expiratory thrust. The result is an emphysema limited to the particular locked air cells. Symptoms often consist of an asthmatic stridor with prolonged expiratory phase.

Stage III: The tumor now occludes and blocks the bronchial lumen. Air previously trapped in the distended alveoli is quickly

taken up by the circulating blood with collapse of these previously distended alveoli. The findings are those of an atelectatic lung segment. Here complications are apt to confuse the findings. Pneumonia, pleurisy with effusion, lung abscess are the complications most frequently encountered.

As a rule, these people are not seen before the stage of atelectasis. X-ray film will confirm the physical findings. Proof of the causative factor can best be obtained by bronchography or laminography.

Differential Diagnosis: Bronchial adenomata usually occur in young adults. With repeated attacks of upper respiratory infection and particularly with hemoptysis the tendency is to consider the case one of pulmonary tuberculosis even though no tubercle bacilli are recovered from sputum or gastric washings.

As there are a number of striking differences between benign adenoma and bronchogenic carcinoma, a table is presented listing their differences.

Diagnosis: These benign polypoid tumors usually arise in the trachea or in one of the larger primary bronchi which can be reached by the bronchoscopist. The adenoma is characteristic as it appears through the bronchoscope. It is a tense, deeply red tumor which is usually covered with a glary mucus. This mucus reflects the light resulting in glistening high lights. On wiping

Bronchial Adenoma	Bronchogenic carcinoma
Infrequent—2 cases admitted to Brooklyn Cancer Institute in 10 years.	Becoming more frequent, 273 cases admitted to Brooklyn Cancer Institute in 10 years.
Occurs in young adults 20-30 years of age.	Occurs in older adults 50-65 years of age.
60 per cent in females (reported cases).	99 per cent in males: our experience in reported cases.
Hemoptysis is apt to be severe and repeated.	Hemoptysis is only a secondarly phenomenon. When bleeding occurs, is usually not copious.
History is that of repeated upper respiratory infection with recovery followed by re-infection. Patients live for many years. Death when it occurs is due to complications. Pain is rare.	History is that of rapid downhill course without interval of improvement. Patients usually die within one year of onset of their symptoms. Pain is frequently the outstanding symptom and is without respite.
Metastases do not occur.	Metastases are the rule.
Bronchoscope—Carina is sharp—tumor appearance tense, glistening, bleeds on touch. Involved bronchus is not fixed.	Bronchoscope—Carina is apt to be blunted—tumor appears granular, is less engorged. Affected bronchus is fixed.
Pathology—cells are of uniform size, occur in nests, mitoses are absent, stroma is highly vascular.	Pathology—varies according to type. Cells vary in appearance, mitoses are frequent.

or touching this tumor it is apt to bleed profusely. Proof of diagnosis must be based on biopsy section.

Its histologic detail is characteristic: the cells are of uniform small size, they occur in nests within a highly vascular stroma. Motitic figures are rarely if ever present. With chronicity cartilage and bone and even bone marrow may develop within this tumor.

But two cases of benign bronchial adenoma have been admitted to the Brooklyn Cancer Institute in the past ten years. In this same interval some 272 cases of bronchogenic carcinoma have been admitted, or one case of benign adenoma to every 136 cases of bronchogenic carcinoma. These two cases are reported in detail because both had adenomata involving other organs which produced complications in their clinical course. The presence of multiple adenomata in both these patients would bring up the concept of a possible inherited weakness in these individuals' ectodermal structures.

CASE REPORTS

H. B. is an adolescent woman of Jewish parentage, 17 years of age who in April 1937 was transferred to the Brooklyn Cancer Institute from a neighboring hospital. She stated that a month previously she had been hospitalized because of cough and high fever. She could remember having had similar attacks of cough and fever over the previous 2 years. On examination there was dullness with diminished breath sounds in the lower right thorax. Instillation of iodized oil confirmed an obstruction in the bronchial branch to the right lower lobe (Fig. 2). A previous bronchoscopic biopsy had been erroneously reported as "bronchogenic carcinoma."

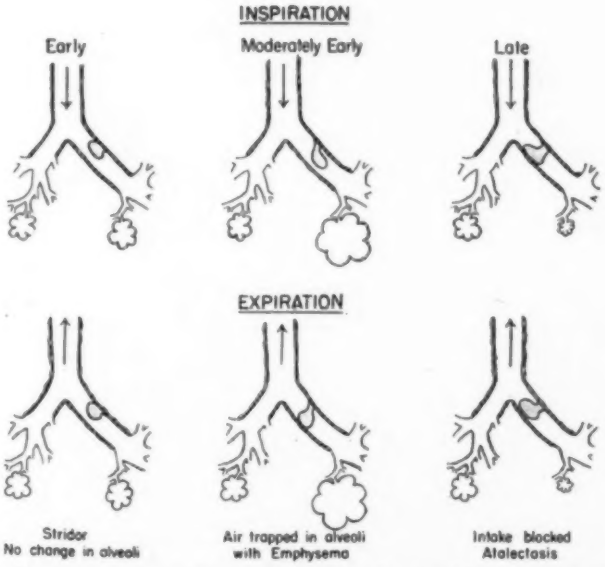

Figure 1: Schematic drawing to demonstrate effect of progressive enlargement of the adenoma on the alveolar structures distally.

She was rebronchoscoped and the diagnosis of benign bronchial adenoma established (Fig. 3). Efforts were made to destroy the adenoma by endoscopic fulguration. Following an acute flareup of the pneumonic infection, she developed an encapsulated empyema. This was drained by resecting a rib. In spite of this, a lung abscess developed behind the obstructed bronchus. After a prolonged convalescence of 6 months she was discharged from the Brooklyn Cancer Institute as improved.

February 1938, one year after her first hospitalization, she was readmitted. At this time, further attempt was made to drain the lung

FIGURE 2

FIGURE 4

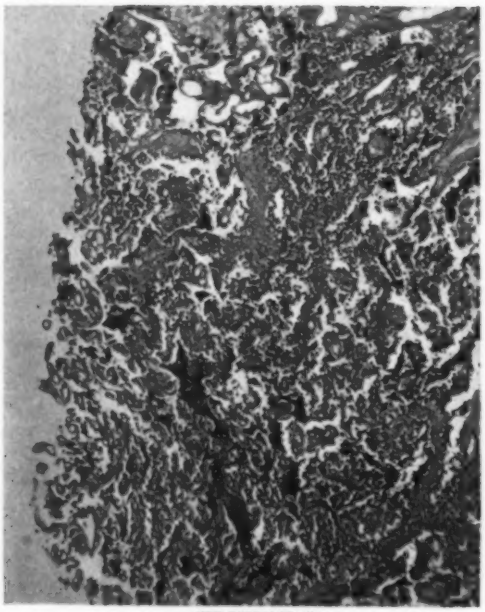

FIGURE 3

Fig. 2: H.B. Iodized oil appears as ring at the site of adenoma plugging the secondary bronchus to the lower right lobe. The atelectatic lower lobe overlies the diaphragm.

Fig. 3: H.B. Benign bronchial adenoma. Nests of small cells of uniform appearance in a highly vascular stroma.

Fig. 4: H.B. Appearance of chest after thoracoplasty and removal of lower and middle lobes. Note the extensive scarring with incomplete expansion of the upper lobe into the lower thorax and appearance of a large residual abscess cavity.

abscess and two large partially encapsulated empyema accumulations. Radiographs demonstrated extensive bronchiectatic cavitation in the diseased lung distal to the plugged bronchus. Clubbing of her fingertips became evident. She was given several small transfusions and by April 1938 the bronchial fistula seemed to be healing and she was discharged again as improved.

In June 1939 (over 2 years after her first hospitalization), she was admitted into another hospital. There an unsuccessful attempt was made to remove the right lower lobe. It was not till August of that year that the surgeon was able successfully to remove the diseased middle and lower lobes (Fig. 3). After a prolonged convalescence she gained weight and appeared symptomless, except for a persistent draining chest sinus which did not close until 1940.

FIGURE 5

FIGURE 6

Figure 5: H.B. Appearance in 1937, before bronchial or pituitary adenomata appeared.—*Figure 6:* H.B. Taken in 1946, showing facial changes due to pituitary adenoma.

Figure 7: Hand (left) before flaring of tufts and coarsening of bone structure and (right) same following changes due to influence of pituitary adenoma.

In 1945 this sinus re-opened and in June 1946 her cough returned and she again began to raise large amounts of purulent, foul smelling sputum (Fig. 4). A large cavity had formed in the retained right upper lobe. These symptoms were partially controlled by large doses of penicillin.

In 1941 she noticed that her lower teeth were spreading, and that her nose and chin were apparently becoming more prominent, her skin was assuming a coarsened texture and abnormal hair appeared on her forearms and over the pubic area (Figs. 5 and 6). One day she accused a shoe salesman of having given her shoes of too small a size; she also noticed that her gloves were too tight to wear (Fig. 7). Her menses ceased. It was, however, not until 3 years later that examination of her

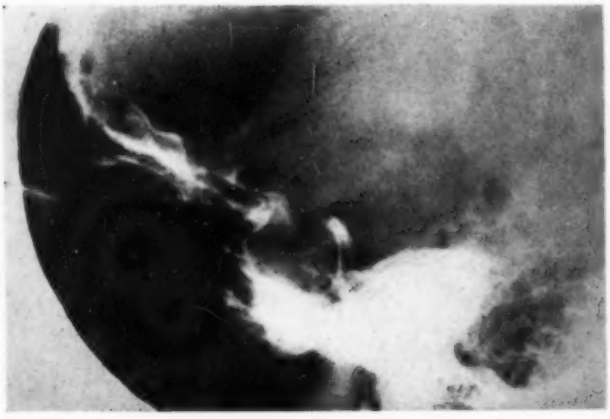

Figure 8: H.B. No. 608. Balooned out sella turcica due to a large eosinophilic granuloma.

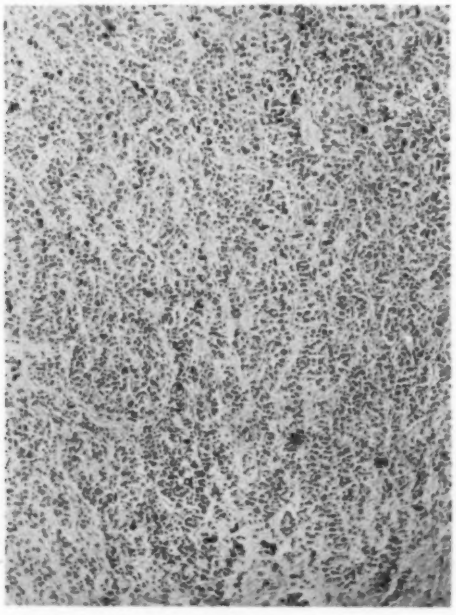

Figure 9: H.B. Pituitary adenoma.

visual fields demonstrated a bitemporal hemianopsia. X-ray film revealed a balooning out of her sella turcica (Fig. 8). With the facts listed above, there is little doubt that this patient also had an eosinophilic adenoma of the pituitary gland (Fig. 9).

C. H. a 50 year old Polish male was first admitted into the Brooklyn Cancer Institute in 1938. He stated that for 22 years (since 1916) he had had "bouts of lung trouble." These attacks consisted of chills, fever with cough and occasionally he coughed up large amounts of bright red blood. Of late, he noticed that he is increasingly "short of breath."

He had been treated in a number of hospitals for pneumonia. Finally

Figure 10: C.H. Large wedged atelectatic mass to the right of the heart—right lower lobe.

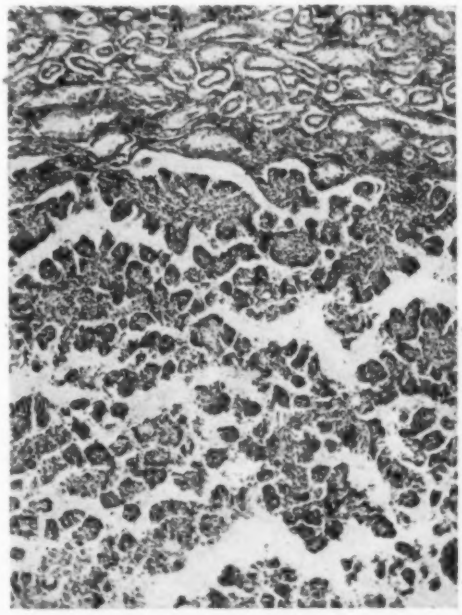

Figure 11: C.H. Kidney lesion. Benign papillary adenoma.

a diagnosis of pulmonary tuberculosis was arrived at. He was sent to a hospital for the treatment of tuberculosis. Later he sought treatment at a tuberculosis sanatorium in Colorado. Since his return from Colorado, he again had several bouts of "pneumonia." During his last hospitalization, a large cystic tumor of his left kidney was discovered and removed. It was during his convalescence from this procedure that he was transferred to the Brooklyn Cancer Institute.

On admission, his temperature fluctuated widely for several days, then gradually subsided. Physical findings were dullness with absent breath sounds but increased vocal fremitus throughout the lower half of the right thorax. The liver, moderately tender could just be palpated. His fingers were clubbed. No tubercle bacilli or actinomycosis rods were recovered on repeated examinations of his sputum.

X-ray film (Fig. 10), demonstrated a triangular opacity extending laterally from the right border of the heart to the costophrenic sinus. This was proven to be an atelectatic right lower lobe. A large amount of pus which kept welling out of the lower right main bronchus made the bronchoscopic examination difficult. After aspiration a tense tumor mass was sighted, this bled profusely on touching. A biopsy was taken and reported as bronchial adenoma.

Treatment: The endobronchial neoplasm was electrocoagulated on several occasions. Each attempt was followed by excessive bleeding. Three months after admission (June 29, 1938) he died as a result of a profuse pulmonary hemorrhage.

In 1937 he had had a melon sized cystic left kidney removed; section

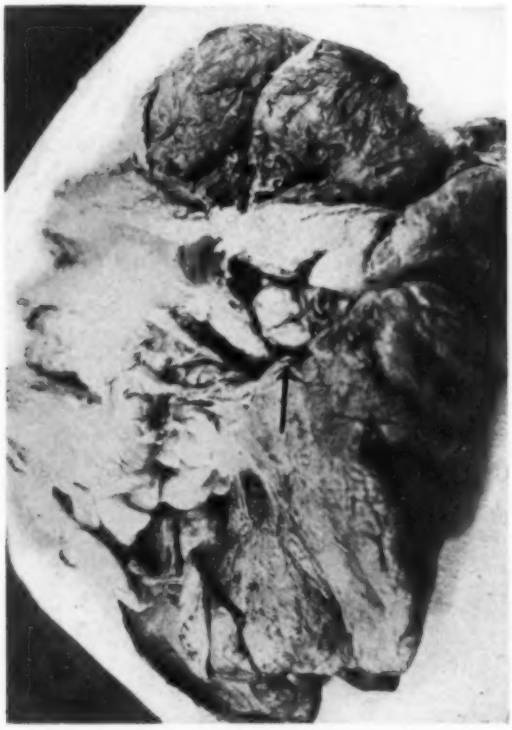

Figure 12: C.H. Right lung demonstrating adenoma filling lower right main bronchus.

of this kidney was never obtained. At necropsy a discrete cortical adenoma was discovered in the remaining kidney (Fig. 11).

Necropsy: A globular yellow white tumor mass plugs the lumen of the lower right main bronchus. This is attached to the bronchial wall by a broad base. Its surface appears necrotic (fulguration). The right main bronchus is partly filled with recent blood clot (Fig. 12).

Microscopic examination: The tumor is composed of groups of cells in alveolar, papillary and row form (Fig. 13). The individual cells are uniform in appearance, have a moderate amount of pink cytoplasm, are cuboidal or polygonal. Nuclei are uniform in appearance, small, round or ovoid and vesicular, occasionally hyperchromatic. There are no mitotic figures. There is absence of any inflammatory cellular infiltration. The mass is completely surrounded by a wall of fibrous tissue. In spots, a regular dense bony structure has been laid down. This has occurred in previous dense fibrous bands. A suggestive marrow is occasionally present containing numerous dilated blood spaces (Fig. 14). The site of diffuse hemorrhage has undergone coagulation necrosis (post operative?).

SUMMARY

The literature of benign bronchial adenoma is reviewed.

This tumor may be mistaken for pulmonary tuberculosis or bronchogenic carcinoma.

Treatment is endoscopic fulguration or lobectomy.

Two cases of bronchial adenoma are reported, interestingly each had adenomatous involvement of other organs.

FIGURE 13 FIGURE 14

Figure 13: C.H. Lung tumor. Typical nests of tumor cells in a rather scant, poorly cellular stroma.—*Figure 14:* C.H. Lung tumor. Formation of bone and marrow within the tumor.

RESUMEN

Se repasa la literatura relativa al adenoma bronquial benigno.

Se puede confundir este tumor con la tuberculosis o el carcinoma broncógeno.

El tratamiento consiste de fulguración endoscópica o lobectomía.

Se informa sobre dos casos de adenoma bronquial en los que se notó con interés que ambos tenían invasiones adenomatosas de otros órganos.

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Bronchiectasis in Children*

ALBERTO CHATTAS, M.D., F.C.C.P.
Cordoba, Argentina

Since the classic description of the bronchiectasis by Laennec in 1819, our knowledge about dilatation of the bronchial tree has greatly increased through:

1) Anatomopathological studies.
2) Bronchographic studies.
3) Endoscopy.
4) Surgical treatment.

Modern bronchographic technic has made easy the early discovery of bronchiectasis. It is also applicable routinely during the first years of life, permitting modern surgical treatment, with very little risk and excellent results even in extensive bilateral cases (Overholt and Langer).[1] Unfortunately, we still too frequently see children harboring bronchiectasis for many years without diagnosis and dying of some broncopulmonary complication.

Reviewing the official mortality statistics in the city of Córdoba, we see that from 1930 to 1940 no case of death caused by bronchiectasis in children under 15 years of age, was reported. No case was reported under the label of chronic bronchitis or nonspecified bronchitis in 1930. In 1935, 4 cases were reported, all of them under 9 years of age (2 chronic and 2 nonspecified); in 1940, 3 cases were reported, all of them under 1 year of age (1 chronic and 2 nonspecified). This lack of figures relative to mortality due to bronchiectasis in children shows obviously, how the diagnosis of this disease in the medical practice is overlooked, even now.

In our city, the third largest in the Argentine, with a population of 325,000 inhabitants, its infant mortality due to pulmonary disease is: 19 per cent of the general mortality from 0-1 year of age; 20.4 per cent of the general mortality from 1-4 years of age; 8.8 per cent of the general mortality from 5-9 years of age.

In the Children's Hospital of Córdoba during the last 10 years, of 27,636 patients, only 60 cases were hospitalized on account of bronchiectasis, that is 0.2 per cent.

It is estimated that in children, bronchiectasis represents about 5 per cent of all diseases. Bonniot,[2] reports that one third of the

*Presented at the 13th Annual Meeting of the American College of Chest Physicians, Atlantic City, 1947.

children with bronchiectasis die in 2-3 years after acquiring the disease. Head[3] states that persons who have developed bronchiectasis during childhood live only to 40 years of age. Perry and King,[4] studying 96 patients with bronchiectasis in the first decade of life, noted that 65 per cent die within 20 years and 90 per cent within 30 years.

Bronchiectasis in Pediatrics

The study of the disease in adults shows the frequency of early symptoms during childhood, an age in which a number of illnesses complicate bronchial changes (especially whooping cough). Laennec said that out of every four bronchiectasis in the adult, three had their origin in childhood. According to other reports, symptoms of bronchiectasis in adults is traceable to the first decade of life in 27 per cent and to the first two decades in 57 per cent.

In the first decades of life, the symptoms of bronchiectasis are intermittent, and often masquarading as acute lung diseases, especially pneumonia. We always remember the old teaching of Czerny[5] who said, "that every child that has repeated acute pneumonia with the same localization of the lesion, should make the physician think of bronchiectasis."

The responsibility of the pediatrician is greater than that of the general practitioner, because he has the primary responsibility in the early recognition of the disease and stands in "the first line of defense" as Brennemann[6] said. The diagnosis can be frequently suspected from the history of the patient and from the physical examination; however a precise diagnosis can only be made as we mentioned before, by bronchographic studies.

Classification of Bronchiectasis in Children

Bronchiectasis is classified as *congenital* and *acquired*. The congenital type was considered till not many years ago, as the most frequent, but new systematic investigation of the bronchial tree shows now that acquired bronchial changes are the most common during the first decade of life. *Congenital* bronchiectasis is produced by a lack of development in the embrionic stage that Castex[7] calls "alveolo-agenesic-bronchiectasis."

Recent researches attribute these congenital malformations to virus diseases, acquired by the mother during the first 3 months of pregnancy (Erickson, Conte and his associates[8,9]). According to the experimental work of Warkany,[10] the malformation in the embryo may be due to a lack of vitamin A. or B. Kartagener[11] published his observations on bronchiectasis in "Situs viscerum inversus" and describe what is known as the "triad of Karta-

gener" that is, the coexistence of bronchiectasis, sinusitis and congenital dextrocardia.

Pathogenesis of Bronchiectasis in Children

Acquired bronchiectasis may result from the following diseases: 1) Bronchial infections, especially whooping cough. 2) Measles. 3) Chronic pneumonitis, sclerosis and lung abscesses. 4) Foreign bodies. 5) Atelectasis. 6) Tuberculosis. 7) Infection of tonsils and paranasal sinuses. 8) Allergic catarrhs.

Especially in children under 5 years of age, after acute or subacute bronchitis, there is a change in the bronchial mucous membrane, with a thickening of it. These changes are also observed after whooping cough, measles and repeated grippes. Following these changes of the mucous membrane, alterations in the basal stratum of the bronchial wall appear, becoming chronic and developing into definitive bronchiectasis. Transitory bronchial enlargement during whooping cough may be due to muscular paralysis of the bronchus in the neighboring zones of the inflamed mucosa. We must remember that histologically, the small bronchi are principally formed by muscles that occupy the greatest part of the bronchial wall and contribute with their tonus to the preservation of the normal bronchial form.

Roentgenologic studies of the chest are of great value in discovering atelectasis in some segments of the lung. Atelectasis sometimes preceeds, coexists or follows a bronchiectasis and it may be due to bronchial obstruction produced by infections (Anspach[12]).

It is important to bear in mind that bronchiectasis involving an entire lobe may be produced by foreign bodies. We shall briefly describe the following cases:

A girl, 11 years of age, came to us with a bronchopulmonary disease which had begun 5 years before. During the later months, she had repeated hemoptyses. She came to the Children's Dispensary of the Instituto de Tisiología de Córdoba (Director Prof. G. Sayago) because her private physician thought of tuberculosis. X-ray film showed a shadow in the right cardiodiaphragmatic angle.

We ruled out tuberculosis because the tuberculin reaction was negative. A lateral x-ray film showed very clearly the presence of a screw in the basal segment of the right lower lobe. Neither the mother nor the child knew of the presence of a screw in the lung. After a careful inquiry, they both remembered that 5 years before, while the girl had whooping cough, she played with a box of screws and nails. After two bronchoscopic sessions, the foreign body was removed: it was a rusty screw, one inch long (Figs. 1 and 2).

Hemoptysis stopped, but the cough persisted. A bronchogram was performed, showing very clearly bronchiectatic changes in the pulmonary segment obstructed by the screw.

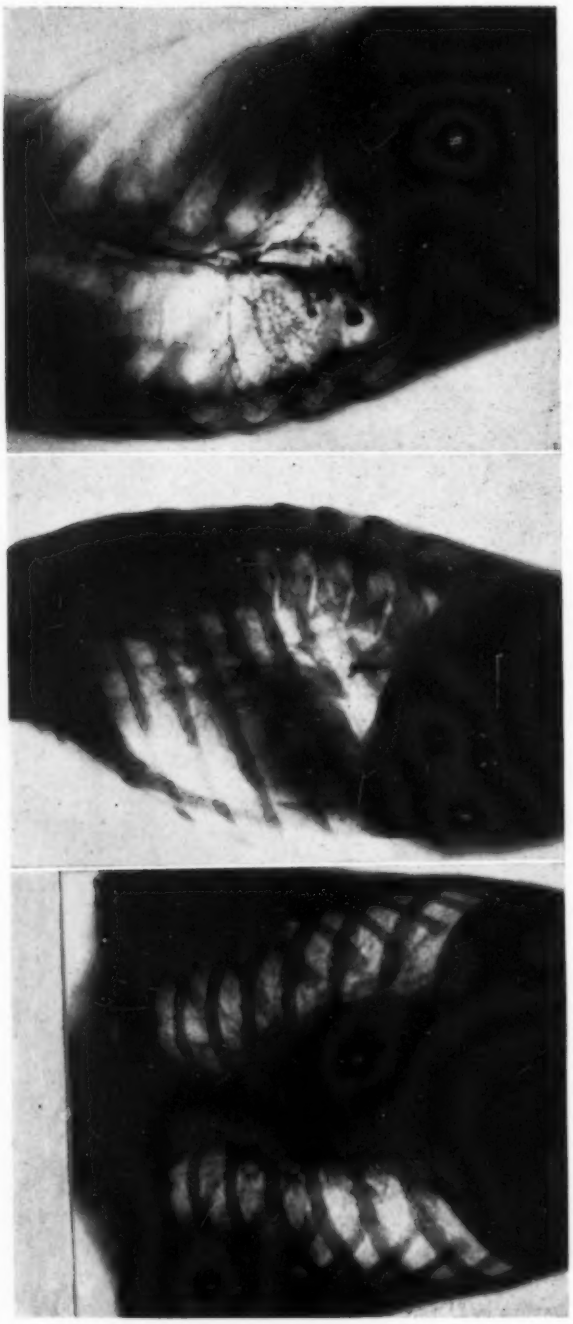

FIGURE 3

FIGURE 2

FIGURE 1

Mycotic infection as a primary disease in the lung may also produce bronchiectasis:

We had such a case: a child, 5 years of age, came to the Children's Hospital (Prof. J. M. Valdes) in very poor condition. She had fever, cough and expectoration for eight months. An x-ray film showed almost complete atelectasis of the entire right lung. Bronchoscopy revealed abundant secretions coming from the right stem bronchus which was narrowed; unfortunately due to his poor condition bronchogram was not done. The child died soon after admission. Post mortem studies revealed typical fungus colony, with bronchiectasis and multiple abscesses (Fig. 4).

Bronchiectasis and Atelectasis in Children

Huizinga[13] of Holland, reporting on 40 cases of bronchiectasis in children, emphasized the importance of atelectasis in producing bronchial alterations. Clinical examinations reveal evidence of a condensed lung. Fluoroscopic or roentgenographic studies will show a triangular shadow, corresponding to an atelectatic segment. Anspach, Ellis, Brennemann, Holinger[6,12,14,15] and others have published several papers dealing with the relationship between bronchiectasis, collapsed lung (shrunken lung) and the triangular shadow seen on the x-ray film. Anspach[12] examining thousands of children's x-ray pictures of the chest found nearly 1 per cent of triangular shadows.

We wish to call attention to the seriousness of atelectasis, especially during the first two years of life, not only because of possible bronchiectasis but also because of secondary infections that may occur. Sometimes the new born suffers from atelectasis, which is not noticeable and which may be the cause of a partly shrunken lung. During the first year of life, thickening of bron-

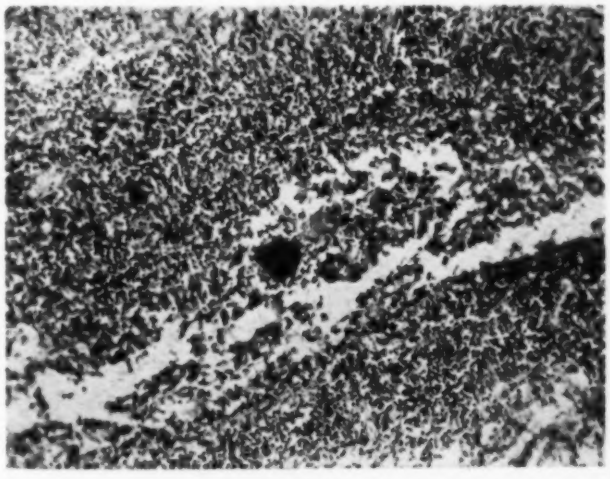

FIGURE 4

FIGURE 7

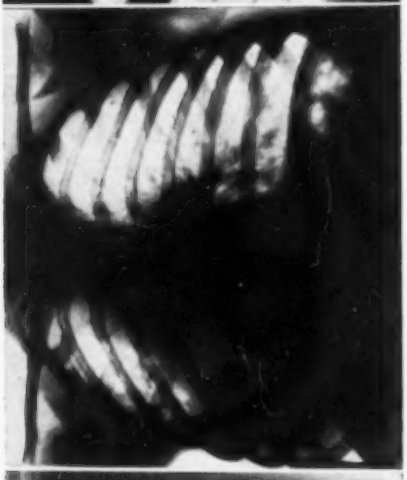

FIGURE 6

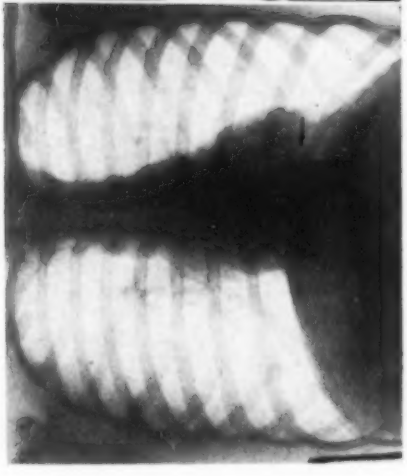

FIGURE 5

chial secretions may sometimes cause obstruction of the small bronchi. This obstruction of the air passage reduces the ingress of air until it causes atelectasis.

We feel that all the broncopulmonary diseases should be followed very carefully by x-ray studies, in different projections. Many small opaque triangular areas can be easily overlooked when the film is taken only in one projection. We pediatricians are responsible to a certain extent for the development of bronchiectasis if we fail to make an accurate diagnosis.

In our practice we have found certain simple measures valuable in preventing or curing atelectasis. We resort to medication that liquify the bronchial secretion; postural drainage, frequent changes of position, naso-tracheal aspiration; and as a last resort bronchoscopic aspiration. In new borns we find that the results obtained by the use of carbogen (95 per cent O_2+5 per cent CO_2) in aerating the lung are very gratifying.

We shall now show the outcome of two cases of atelectasis developed in early life, not diagnosed until complications developed:

A girl, 9 years old, had whooping cough when five years of age and measles at the age of six. For the last 7 years she complained of productive cough. X-ray picture showed a basal triangular shadow beneath the cardiac area (Fig. 5). Bronchographic studies were not done at that time. Operative specimen revealed a small fibrotic and shrunken lower lobe with bronchiectatic changes (Children's Hospital, Prof. J. M. Valdes).

A girl, 6 years of age, had bronchitis when 6 months old and at three years of age had whooping cough. Plain x-ray picture (taken when she came to the Hospital, Surgical Department, Prof. Dr. J. M. Allende), showed an opaque zone in the lower half of right lung with a marked displacement of the heart towards the affected side (Fig. 6). The bronchogram revealed saccular bronchiectasis in the right upper lobe (Fig. 7). The parents refused surgical intervention.

SUMMARY AND CONCLUSION

1) *Atelectasis* is frequent in childhood and must be considered as an important cause of bronchiectasis.

2) It is the duty of the pediatrician to resort to all the diagnostic means at our disposal to make an early diagnosis.

3) *Bronchiectasis* in the adult is a process which frequently originates in the first or second year of infancy.

4) Bronchial changes can take place in whooping cough and measles. These changes may not be permanent, and in this prebronchiectasic stage, medical treatment can be sometimes successful.

5) If medical treatment fails, bronchiectasis becomes defin-

ately established and irreversible; only by surgical treatment can a permanent cure be obtained.

6) Surgical treatment must not be postponed. Children tolerate the intervention better than adults.

7) Our experience deals with 10 cases, 4 boys and 6 girls; between 4 and 13 years of age; 7 were operated upon, and 3 refused operation. Of the 7 operated cases, bronchiectasis was present in 4 in the left lower lobe and in 3 in the right lower lobe. They all made complete recovery; one of the nonoperated patients died. One of the cases showed the triad of Kartagener. In all cases the bronchiectasis was localized in the lower lobe, with exception of one that had bronchiectasis in the right upper lobe, and in one, the bronchiectasis of the left lower lobe was associated with bronchiectasis of the lingula.

RESUMEN Y CONCLUSION

1) La *atelectasia* es frecuente en la niñez y debe ser considerada como una causa importante de bronquiectasia.

2) El especialista en pediatría tiene el deber de valerse de todos los medios de diagnosticar a su disposición a fin de hacer un diagnóstico temprano.

3) La *bronquiectasia* en el adulto es un proceso que frecuentemente se origina en el primero o segundo año de la infancia.

4) En la tos ferina y la alfombrilla pueden ocurrir alteraciones bronquiales que no sean permanentes y en esta etapa pre-bronquiectásica el tratamiento médico es a veces eficaz.

5) Si fracasa el tratamiento médico, la bronquiectasia queda establecida definitivamente y es irreversible, solamente mediante el tratamiento quirúrgico puede obtenerse una curación permanente.

6) No debe aplazarse el tratamiento quirúrgico. Los niños soportan la intervención mejor que los adultos.

7) Nuestra experiencia incluye 10 casos: 4 niños y 6 niñas, de 4 a 13 años de edad. Se operó a 7, tres rehusaron la operación. De los 7 casos operados la bronquiectasia afectaba el lóbulo inferior izquierdo en 4, y el lóbulo inferior derecho en 3. Todos los operados se repusieron por completo, mientras que uno de los pacientes no operados murió. Uno de los casos manifestó la terna de Kartagener. En todos los casos la bronquiectasia estaba localizada en el lóbulo inferior, con la excepción de uno que tenía bronquiectasia en el lóbulo superior derecho y otro en el que la bronquiectasia del lóbulo inferior izquierdo estaba asociada con bronquiectasia del proceso lingual.

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Present Status of Streptomycin In Tuberculosis

KARL H. PFUETZE, M.D., F.C.C.P.*
Cannon Falls, Minnesota

WILLIAM M. ASHE, M.D.**
Rochester, Minnesota

Streptomycin has become established as a valuable weapon in the therapy of tuberculous infections. Tuberculosis is a protein disease, its symptomatology and prognosis variable. As is well known, spontaneous healing may occur without benefit of presently accepted therapeutic measures. Hence it is readily apparent that the final evaluation of any tubercu!o-chemotherapeutic agent will be possible only after long and arduous experimental and clinical study.

Nearly four and one-half years have passed since streptomycin was first reported by Schatz, Bugie and Waksman.[1] This antibacterial agent is produced by certain strains of the actinomycete, Streptomycin griseus. It possesses selective activity against a variety of pathogenic organisms in vitro, among them human and bovine strains of Mycobacterium tuberculosis.

Within a year following the discovery of streptomycin Feldman and Hinshaw[2,3] had found that the antibiotic exhibits a unique suppressive action against tuberculous infections, both experimentally and clinically. This work has been confirmed by many other investigators. Following the early investigations at the Mayo Clinic and Mineral Springs Sanatorium, various Federal agencies in cooperation with the drug companies producing streptomycin instituted a centralized program of experimental and clinical research. As a consequence, in the past three years many hundreds of carefully selected cases of various forms of tuberculosis have been treated, and many problems inherent in the chemotherapeutic assault on tuberculosis have been clarified. But the complete role of streptomycin in the treatment of tuberculosis has yet to be determined.

Streptomycin in Experimental Tuberculosis

The antibiotic action of streptomycin in experimentally induced tuberculosis in guinea pigs has been well established.

In 1944, Feldman and Hinshaw demonstrated the effectiveness of streptomycin in vivo. In a series of extensive experiments[4] they

*Medical Director, Mineral Springs Sanatorium, Cannon Falls, Minn.
**Fellow in Surgery, Mayo Foundation, Rochester, Minnesota.

proved that streptomycin has the striking ability, not only to inhibit the progress of tuberculous lesions, but also to reverse the potentially fatal course of experimentally induced tuberculosis in the highly susceptible guinea pig. The drug was found to have a relatively low toxicity.

In the fall of 1944, it was felt that cautious use of streptomycin was justified in human beings. Cases selected for the first clinical trials were those in which the prognosis was considered hopeless, such as miliary and meningeal forms of tuberculosis, and nearly terminal pulmonary cases. Careful pharmacological studies for toxic manifestations were made on these patients.[5] The encouraging clinical results obtained and the relatively low toxicity which occurred soon made it apparent that the drug could be used in patients with a more favorable prognosis. A summary of observation on 100 cases of human tuberculosis treated with streptomycin was published in November, 1946.[6]

Clinical Use of Streptomycin

Pulmonary Tuberculosis: In the chemotherapy of pulmonary tuberculosis, it is paramount that there be an intensive evaluation of the individual case prior to commencing treatment. At present, due to the phenomenon of acquired bacterial resistance, it is felt that streptomycin cannot be expected to exhibit its suppressive action for more than one period of treatment. Caution, then, with regard to the long term management of the patient is imperative, because there may be greater need for the use of streptomycin at a future time. Experience to date indicates that the drug is not as effective in many cases of pulmonary tuberculosis as in some other forms of the disease.

The maximum therapeutic benefit of streptomycin in pulmonary tuberculosis occurs in pneumonic and early exudative lesions. There may be some degree of improvement in proliferative lesions, but the pathologic tissue changes occurring in fibrocaseous and fibrocavernous lesions are often irreversible. In minimal pulmonary tuberculosis streptomycin therapy is seldom indicated.

With some exceptions, the selection of cases to receive streptomycin is best determined by roentgenographic and clinical observation over a period of at least several weeks. Results will be more nearly comparable when there has been an adequate trial of rest with or without other therapeutic procedures. A lesion that has been progressive or has remained stationary with previous therapy may respond favorably to the retardative action of streptomycin, permitting the natural defense mechanisms of the host to accelerate the reparative processes.

The Streptomycin Committee of the Veterans Administration[7]

reported in December 1947, the results of 223 cases of pulmonary tuberculosis treated with the drug under carefully controlled conditions. Of these 223 cases, 156 (70 per cent) were far advanced, 65 (29.1 per cent) were moderately advanced and 2 (0.9 per cent) were minimal. The patients were predominately white males, less than 30 years of age, and were selected because they had progressive or stationary lesions after two months of observation. They received a daily dose of 1.8 grams of streptomycin (0.3 grams every four hours) for 120 days. On the basis of roentgenographic changes, 85 per cent were judged as improved, 10 per cent as unchanged, and 0.5 per cent (1 case) showed progression during the period of treatment. Of 182 cavities at beginning of treatment, 47 (26 per cent) were closed or had disappeared and 67 (37 per cent) were smaller. The remainder were unchanged or larger.

In this study 195 patients were followed by serial roentgenograms for an average of 117 days after cessation of treatment. Of these 40 per cent continued to show improvement, 44 per cent remained unchanged and 16 per cent had extensions or spreads that occurred mainly within 30 days after discontinuing treatment. The authors concluded that "the case for streptomycin rests primarily on the fact that, during treatment, some degree of roentgenographic clearing occurred in 85 per cent of the lesions, of which a majority had been stable or progressive before streptomycin was instituted." They also re-emphasized the fact that streptomycin is an adjunct in the treatment of tuberculosis and superior in suitable cases to bed rest alone.

Similar results are reported by Muschenheim, McDermott, et al[8] in their study of 43 patients, in which they used 3 grams of streptomycin in their early cases for 120 days and one gram daily in 10 cases for 42 days. They concluded that there was some measurable improvement in all of these 43 cases, as shown by roentgenographic clearing.

Results indicate that with the use of streptomycin the scope and frequency of surgical procedures in the treatment of pulmonary tuberculosis are definitely increased. Such procedures are frequently made possible by resolution of exudative lesions, especially in the contralateral lung, and by improving the patient's general condition.

In the selection of cases to receive streptomycin therapy it must be borne in mind that there exists a real hazard of the dissemination of drug-resistant organisms. When patients discharging such organisms leave a sanatorium against advice, they constitute a particularly dangerous public health menace. Experience to date indicates that drug-resistant tubercle bacilli

transferred to previously uninfected individuals will retain their resistance: streptomycin probably will be of no value in the treatment of these people.

Tuberculous Empyema: Streptomycin appears to have little value in the treatment of well established tuberculous empyema. Experience with the drug in this condition has thus far proved to be disappointing. It remains to be seen whether the use of streptomycin in combination with detergents and other antibacterial agents will achieve better results.

Disseminated Tuberculosis: Conclusive evidence of the efficacy of streptomycin therapy is apparent in the unprecedented clinical and objective improvement which often occurs within one to three weeks in the otherwise highly fatal forms of disseminated tuberculosis. Since there is no other known treatment capable of bringing about remission, treatment with streptomycin is mandatory in every case. It is imperative that the disease be recognized early and the maximal tolerated doses of streptomycin be employed immediately, attempting to avoid only the more serious and permanent types of toxicity. In cases of meningitis, treatment with streptomycin should be instituted whenever presumptive evidence indicates that the tubercle bacillus may be the etiological agent, without waiting for bacteriological confirmation.

In tuberculous meningitis the ultimate mortality probably will not be decreased appreciably, but the remission due to streptomycin is often remarkable in degree and may persist for many months. Lincoln and her co-workers[9] report success in the treatment of tuberculous meningitis, using promizole, a sulfone compound, in addition to streptomycin administered intramuscularly and intrathecally. However, her series of cases is small, treatment was begun very early in the course of the disease, and insufficient time has elapsed since termination of treatment to determine whether this combination of therapeutic agents is superior to streptomycin alone.

In generalized miliary tuberculosis the probability of permanent arrest is considerably greater than in tuberculous meningitis or in disseminated tuberculosis with meningeal involvement. Miliary pulmonary lesions may show improvement after only three or four weeks of treatment, and frequently such lesions are scarcely detectable roentgenographically after eight weeks of therapy. They may be complete remission as observed clinically, bacteriologically, and roentgenologically.

Unfortunately the remission which occurs in miliary tuberculosis is not always sustained. It has been shown that streptomycin does not penetrate the substance of the brain and this may be a significant factor in cases which terminate fatally. Not uncom-

monly meningeal involvement becomes manifest when the patient with miliary tuberculosis is already receiving streptomycin intramuscularly in adequate doses. The meningitic process may or may not undergo remission when intrathecal administration of streptomycin is instituted.

When disseminated tuberculosis recurs after a streptomycin-induced remission, a second course of treatment is beneficial only if the infecting organisms are still predominantly sensitive to the drug. In most cases, further treatment is of no value, or the remission effected is less striking in degree and duration than the original remission.

Other Forms of Tuberculosis: Ulcerative and granulomatous lesions of the larynx and tracheobronchial tree respond to streptomycin therapy with almost uniform healing. Improvement may be noted within two weeks during therapy, and in the majority of cases healing usually occurs within six to eight weeks. Administration of the drug is effective by the intramuscular route and relatively ineffective by acrosol therapy alone.

In tuberculous lesions of the oropharynx, streptomycin has proved to be the treatment of choice. The majority of these lesions can be expected to heal completely within two to three months, with doses of one gram daily or less. In one case under our observation, a lesion of the tongue proved tuberculous by biopsy was associated with fibrocaseous pulmonary tuberculosis and positive sputum. Healing of the lesion occurred after six weeks of treatment with a dose of 0.5 gram of streptomycin given intramuscularly in one injection daily.

In tuberculous enteritis streptomycin is the treatment of choice. Cases usually respond with a marked decrease in symptoms within two weeks.

Draining cutaneous sinuses from tuberculosis of the bone, joint, cartilage, and lymph glands respond to streptomycin with rapid and consistent healing. The response in this form of tuberculosis is valuable as it affords an easily objective study of the effectiveness of streptomycin. There is a tendency in a few cases for subsequent recurrence of drainage and enlargement of lymph glands. The Veterans Hospitals have reported that closure of draining sinuses is acclerated by removal of pus and necrotic material surgically.

Tuberculosis of bone and joints appear to be retarded in some cases sufficiently to produce healing, alone or in conjunction with orthopedic surgery.[10] Further study is necessary to permit conclusions as to the ultimate place of streptomycin in treatment in these conditions.

Genito-urinary tuberculosis has responded to streptomycin,

chiefly by improvement in symptoms. The drug is of value as an adjunct when surgery is indicated and is the treatment of choice when surgery is impossible, as in tuberculosis of a solitary kidney, or bilateral renal tuberculosis. Its palliative effects are pronounced and prolonged in more than 50 per cent of the lesions treated.[11] At the Veteran's Hospital at Bronx,[12] New York, 23 out of 32 cases of tuberculosis of the genito-urinary tract were reported as improved, but the investigators draw no conclusions as to the maintenance of improved status.

In tuberculous peritonitis, the number of cases treated with streptomycin has been small, but reports indicate that the drug has a definitely favorable effect on the course of the disease. It appears that streptomycin is the treatment of choice in such cases, combined with rest and other accepted therapy.

Tuberculous pericarditis, to date, has not responded successfully to streptomycin in the great majority of cases. This may be due to failure in starting treatment sufficiently early in the course of the disease.

The response of the various forms of skin tuberculosis to streptomycin has been variable, with a tendency toward recurrence of the lesions.

Evidence accumulated thus far would indicate that streptomycin is often beneficial in tuberculous otitis media. We believe it will prove to be an important factor in the treatment of such cases, combined with other accepted therapy.

In ocular tuberculosis, the number of cases reported to date, treated by streptomycin, is too small to permit drawing any conclusions.

Administration and Dosage

For all forms of tuberculosis except meningitis, streptomycin should be given parenterally, preferably by intramuscular or deep subcutaneous injection. In tuberculous meningitis, daily intra thecal injections of 25 to 100 milligram in addition to the intramuscular administration were previously considered imperative. However, this opinion has been challenged recently by some investigators both in the United States and in Europe. Investigations carried out at Herman Kiefer Hospital in Detroit have led the group there to believe that intrathecal injection of streptomycin has no particular value and is contra-indicated because of the severe toxic manifestations which may result from this method of administration.[13] Undoubtedly, further clinical study will determine which of these two views is correct. In any event, early diagnosis and treatment is most essential to recovery. A minimal dosage of two grams daily, given intramuscularly, for at

least three to four months is recommended at this time for this disease.

For all other forms of tuberculosis, a total dose of one gram daily for two to three months is sufficient. Some investigators are reporting good results in many cases with doses of 0.5 gram daily or less for periods of 30 to 60 days. Frequency of administration may vary from 12 to 24-hour intervals, apparently with similar results clinically. The present trend seems to be toward a single injection daily. The optimal duration of therapy will vary considerably in different cases, depending upon the time of appearance of streptomycin-resistant strains. Further investigations will undoubtedly clarify the problems of daily dosage, frequency of administration, and duration of treatment.

Toxicity: Most investigators have been impressed with the relative lack of serious toxic effects from streptomycin. The potential toxicity should not be a deterrent when indication for treatment is definite.

The three chief toxic manifestations of streptomycin are: (1) damage to the eighth nerve resulting in vestibular disturbance, (2) renal irritation, and (3) sensitization reactions. Vestibular disturbance is noted in the large majority of patients who receive 2 or more grams daily for extended periods of time. Dizziness and ataxia may vary in severity and in time of appearance. They may occur early or late in the course of treatment. The loss or decrease of labyrinthine function can be demonstrated by caloric stimulation tests, and this loss appears to be permanent.

Fortunately most patients can compensate for vestibular dysfunction to a remarkable extent. A number of our patients, in whom there is complete loss of vestibular function as demonstrated by caloric stimulation tests, have returned to their former occupation. Middle aged and elderly patients compensate more slowly and to a less degree than younger individuals.

The danger of deafness from treatment with streptomycin is neglible. Cases in which a partial loss of hearing has been reported have occurred only when concentration of streptomycin in the blood were excessive, due either to extremely high dosage or to renal insufficiency. In such cases hearing is restored if the drug is discontinued promptly. It is interesting to note that cases of permanent deafness which have been reported have occurred only in patients with tuberculosis meningitis.

Cylindruria and low grade albuminuria occur not infrequently during treatment with streptomycin. It is now accepted that permanent renal damage referable to streptomycin has occurred only when there was evidence of renal impairment before treatment.

Sensitization reactions may occur occasionally after treatment of one to three weeks, with manifestations of chills, fever, nausea, and skin rash. In such cases the drug should be discontinued until these symptoms have subsided. Treatment may then be resumed with minute doses of 0.1 gram daily, gradually increasing the dosage over a period of two to four weeks until the desired daily dosage is reached.

Isolated cases of serious toxic effects from streptomycin have been reported by the army, navy, and Veteran's Administration,[14] including three cases of exfoliative dermatitis, one case of agranulocytosis, and two cases of aplastic anemia in which the drug is suspected of being a factor. Fortunately such cases are rare.

The toxicity of streptomycin parallels the dosage and, to a lesser extent, the duration of treatment. Among patients receiving two grams daily for three to four months, mild to severe vestibular disturbance as determined by caloric stimulation tests, can be anticipated in 90 per cent or more. In our experience, when doses of one gram daily for similar periods are used, the toxic manifestations with reference to the vestibular mechanism are markedly reduced. Approximately one half of these patients revealed no vestibular dysfunction by caloric stimulation tests.

Bacterial Resistance: The phenomenon of bacterial resistance to streptomycin is the major limitation to the use of this drug in the treatment of tuberculosis. Evidence has accumulated to indicate that in approximately three-fourths of the cases the bacterial population is predominantly resistant after two to four months of treatment. In most cases this resistance has proved to be permanent. Studies have shown that resistance may occur as early as one month after treatment is started. The importance of this problem is obvious, since further treatment with streptomycin has proven to be ineffective when the infecting organisms are drug-resistant.

It is hoped that some method will be found to prevent or delay the occurrence of resistance. Perhaps some other anti-tuberculosis agent may be discovered which is clinically applicable and can be used alone or in combination with streptomycin to achieve this end.

SUMMARY

Streptomycin is now accepted as a valuable weapon in the treatment of tuberculosis. Like other valuable drugs, it has its assets and limitations. It can not be considered a substitute for sanatorium care and other well established measures such as collapse therapy. It is a "must" in the treatment of (1) miliary tuberculosis, (2) tuberculous meningitis, (3) ulcerative tracheobronchial tuberculosis, (4) tuberculosis of the larynx and oro-

pharynx, (5) draining cutaneous sinuses, and (6) tuberculous enteritis. In tuberculous peritonitis it is probably the treatment of choice. In genito-urinary tuberculosis its value is chiefly palliative and as an adjunct to surgery. In bone and joint tuberculosis it is helpful in some cases, both alone and in conjunction with surgery.

In pulmonary tuberculosis it is a valuable adjunct in exudative and pneumonic lesions, in combination with bed rest and collapse therapy, or resection when indicated. It must be emphasized that an understanding of the pathology of tuberculosis and of the mechanism of anti-bacterial therapy is necessary if the best possible end results are to be obtained by use of the drug. Streptomycin has proved that tuberculosis is amenable to chemotherapy. It is fervently hoped that other and even more effective agents may be discovered.

RESUMEN

Actualmente se reconoce que la estreptomicina es una arma valiosa en el tratamiento de la tuberculosis. Lo mismo que otras drogas valiosas, tiene sus ventajas y sus restricciones. No puede ser considerada como substituto de la atención sanatorial o de otras medidas bien establecidas, tales como la colapsoterapia. Es obligatorio que se le emplee en el tratamiento de (1) la tuberculosis miliar, (2) la meningitis tuberculosa, (3) la tuberculosis tráqueobronquial ulcerativa, (4) la tuberculosis de la laringe y de la faringe bucal, (5) las fístulas cutáneas y (6) la enteritis tuberculosa. En la peritonitis tuberculosa es probablemente el tratamiento preferible. En la tuberculosis génitourinaria su valor es principalmente paliativo y como coadyuvante de la intervención quirúrgica. En la tuberculosis de los huesos y las articulaciones es útil en algunos casos, tanto sola como combinada con la intervención quirúrgica.

En la tuberculosis pulmonar es un coadyuvante valioso en lesiones exudativas y neumónicas, combinada con el reposo en cama y la colapsoterapia, o la resección cuando esté indicada. Debe recalcarse que es necesario comprender la patología de la tuberculosis y el mecanismo de la terapia antibacterial para que se puedan obtener los mejores resultados posibles con el empleo de la droga. La estreptomicina ha demostrado que la tuberculosis es tratable mediante la quimioterapia. Se espera fervorosamente que se puedan descubrir otros agentes aún más eficaces.

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Editorial

ANOTHER OUTSTANDING PROGRAM

The Program Committee, of the American College of Chest Physicians, with Dr. Richard Overholt as Chairman, has arranged for a splendid array of speakers on a large variety of subjects pertaining to diseases of the chest for the Fourteenth Annual Meeting of the College to be held at the Congress Hotel in Chicago from June 17 to 20, inclusive. A copy of the preliminary program appears in this issue of "Diseases of the Chest." Many nations are represented by speakers on this program. Thus there will be an opportunity for physicians from various parts of the world to compare diagnostic criteria and preventive measures as practiced in their nations. It has long been recognized that most of the problems in chest diseases must be solved on a world-wide basis. This program, therefore, affords a marvelous opportunity to exchange thoughts, ideas and methods leading to coordination of activity throughout the world. In addition to the formal addresses and discussions, numerous clinics and demonstrations will be held.

This year a new feature of round table luncheon meetings has been added to the program of the College. Here all physicians who desire will have an opportunity to discuss and contribute to the knowledge of the subjects under consideration at the various round table sessions.

At the annual meeting of the American Medical Association held in Atlantic City in 1947 a Section on Diseases of the Chest in the Scientific Assembly of the American Medical Association was established. This was a far-reaching step toward the goal of chest disease control and eradication. The first program of this newly created Section on Diseases of the Chest will be held on Thursday morning, June 24th. This will consist of a symposium on streptomycin presented by Drs. Bogen, Hinshaw, Pfuetze, Tucker, Beattie and Keefer, all of whom have done outstanding work on various aspects of the therapeutic uses and values in tuberculosis. It is exceedingly important that all members of the College of Chest Physicians, after their regular session, remain for the meetings of the American Medical Association and be present for the program of the newly created Section on Diseases of the Chest.

It is anticipated that the attendance at the Chicago meeting of the College in 1948 will exceed that of the Atlantic City meeting in 1947 which was the largest attendance on record. A great deal of time has been devoted to the preparation of the program, and the speakers and discussers are putting forth their best efforts on a large volume of material covering nearly all phases of chest diseases that will be interestingly presented, highly informative and thoroughly practical. Thus, every physician who attends these sessions can become better equipped to practice chest diseases when he returns to his home sphere of activities.

J. A. M.

FOURTEENTH ANNUAL MEETING

AMERICAN COLLEGE OF CHEST PHYSICIANS

CONGRESS HOTEL, CHICAGO, ILLINOIS

June 17 - 20, 1948

P R O G R A M

Thursday, June 17

Oral and Written Examinations for Fellowship

The Board of Examiners of the College will conduct oral and written examinations for Fellowship on Thursday, June 17, at the Congress Hotel, Chicago, Illinois. The members of the Board of Examiners are:

Alvis E. Greer, M.D., Houston, Texas, Chairman
Edward W. Hayes, M.D., Monrovia, California, Vice-Chairman
Edgar Mayer, M.D., New York, New York, Secretary
Louis H. Clerf, M.D., Philadelphia, Pennsylvania
Alton Ochsner, M.D., New Orleans, Louisiana

Executive Council Meeting

Board of Regents Meeting

Board of Governors Meeting

Council and Committee Meetings

Friday, June 18

9:00 a.m. SESSION NO. I

Dr. Edwin R. Levine, Chicago, Illinois, Moderator

Dr. Irving Sarot, New York, N. Y.: "Enucleation Technique for Lung Containing Adhesions"
Discussor: Dr. William Tuttle, Detroit, Michigan

Dr. Francisco Torres, Cordoba, Argentina: "Pulmonary Resection in Tuberculosis"
Discussor: Dr. J. D. Murphy, Oteen, North Carolina

Dr. George Wright, Saranac Lake, New York: "Effect of Disease on Pulmonary Physiology"

Dr. George G. Ornstein, New York, N. Y.: "Pulmonary Function"
Discussor: Dr. Benjamin P. Potter, Jersey City, N. J. (To discuss both Dr. Wright's and Dr. Ornstein's papers)

2:00 p.m. SESSION NO. II

Dr. Richard H. Overholt, Brookline, Massachusetts, Moderator

Dr. Karl Poppe, Portland, Oregon: "Treatment of Aortic Aneurysms by Wrapping with Foreign Body"
Discussor: Dr. Osler Abbott, Atlanta, Georgia

Dr. Charles P. Bailey, Philadelphia, Pennsylvania: "Surgical Treatment of Mitral Stenosis"
Discussor: Dr. Horace G. Smithy, Charleston, South Carolina

Dr. Willis J. Potts, Chicago, Illinois: "Recent Advances in Intrathoracic Vascular Surgery"

Dr. O. T. Clagett, Rochester, Minnesota: "Surgical Treatment of Emphysematous Blebs and Bullae"
Discussor: Dr. Francis M. Woods, Brookline, Massachusetts

Dr. Evarts A. Graham, St. Louis, Missouri: "Problem of Cancer of the Lung"
Discussor: Dr. W. E. Adams, Chicago, Illinois

Saturday, June 19

9:00 to 10:30 a.m. ADMINISTRATIVE SESSION

Recommendations for elective officers for the College should be sent to the Chairman of the Nominating Committee:

Walter E. Vest, M.D., Huntington, West Virginia, Chairman
Carl H. Gellenthien, M.D., Valmora, New Mexico
Hubert A. Boyle, M.D., New Bedford, Massachusetts

10:30 a.m. SESSION NO. III

X-ray Conference (Proved cases of unusual interest will be discussed)

2:00 p.m. SESSION NO. IV

Dr. Francis J. Weber, Washington, D. C., Moderator

Symposium on BCG Vaccination

Dr. Ismael Cosio Villegas, Mexico City, Mexico: "The Experience with BCG Vaccination in Latin America"

Dr. Milton I. Levine, New York, N. Y.: "Experience with BCG Vaccination in New York City"

Dr. Jose I. Baldo, Caracas, Venezuela: "BCG Vaccination in Venezuela"

Dr. Gumersindo Sayago, Cordoba, Argentina: "Experience with BCG in Argentina"

Dr. Arlindo de Assis, Rio de Janeiro, Brazil: "Concurrent BCG Vaccination"

Dr. Herman E. Hilleboe, Albany, New York: "The BCG Program of New York State"

"Closing Remarks," Chairman

Sunday, June 20

9:00 a.m. SESSION NO. V

Dr. Andrew L. Banyai, Milwaukee, Wisconsin, Moderator

Dr. Marcio Bueno, New Bedford, Massachusetts: "Diagnostic Bronchial Lavage in Tuberculosis"

Dr. George N. Papanicolaou, and Dr. Henry A. Cromwell, New York, N. Y.: "Diagnosis of Cancer of the Lung by the Cytologic Method"

Dr. Seymour M. Farber, San Francisco, California: "Diagnosis of Primary Carcinoma of the Lung by means of Cytological Examination of Sputum and Bronchial Secretions"

Dr. Maurice M. Black, Brooklyn, New York: "Biochemical Studies in Cancer Diagnosis"

Dr. Andre Soulas, Paris, France: "Study of Bronchial Stenosis in Bronchopulmonary Tuberculosis"

2:00 p.m. SESSION NO. VI

Dr. Harry C. Warren, San Francisco, California, Moderator

Dr. Allan Hurst, Dr. Jules V. Coleman, and Ruth Hornbein, M.S.S., Denver, Colorado: "The Place of Psychiatry in the Problem of a Tuberculosis Hospital"

Dr. Willard Van Hazel, Dr. Paul H. Holinger, and Dr. Robert J. Jensik, Chicago, Illinois: "Adenoma of the Bronchus"

Dr. Italo Volini, Dr. James R. Hughes, and Dr. John Peffer, Chicago, Illinois: "A Comparative Study of Sulfadiazine, Penicillin and Penicillin Combined with Sulfadiazine in the Treatment of Lobar Pneumonia"

Dr. Ben E. Goodrich, and Dr. T. D. Johnson, Detroit, Michigan: "Chronic Bilateral Basal Pulmonary Fibrosis"

Dr. Maurice Segal, Boston, Massachusetts: "Facts and Fancies in the Management of the Seriously Ill Patient with Bronchial Asthma"
Discussor: Dr. George L. Waldbott, Detroit, Michigan

Note: The names of the additional discussors will appear in the final program

COUNCIL LUNCHEON MEETINGS

Thursday, June 17

Annual Conference of College Chapter Officials:

Dr. Carl C. Aven, Atlanta, Georgia, Chairman, Presiding

Dr. Seymour M. Farber, San Francisco, California, Secretary-Treasurer, California Chapter: "Report of the Conference Secretary"

Dr. Nelson W. Strohm, Buffalo, New York, Past-President, New York State Chapter, Vice-Speaker, House of Delegates, New York State Medical Society: "Preparing and Presenting a Program on Diseases of the Chest in Connection with the Annual Meeting of the State Medical Society"

Dr. Irving Willner, Newark, New Jersey, Past-President, New Jersey Chapter: "Establishing a Section on Diseases of the Chest in a State Medical Society"

Dr. Leon H. Hirsh, Milwaukee, Wisconsin, Secretary-Treasurer, Wisconsin Chapter: "Program Planning and Attendance Building for Chapter Meetings"

Dr. W. Bernard Yegge, Denver, Colorado, Secretary-Treasurer, Rocky Mountain Chapter: "Correlating Chapter Activities with the Program of the National Organization"

Dr. Donato G. Alarcon, Mexico City, Mexico, President, Mexican Chapter: "Chapters in Other Countries"

Friday, June 18

Conference of Medical Directors and Superintendents of Tuberculosis Hospitals and Sanatoria:

Dr. R. S. Anderson, Erie, Pennsylvania, Chairman, Presiding; Dr. E. W. Custer, South Bend, Indiana, Secretary

Dr. Paul A. Turner, Medical Director, State Tuberculosis Sanatorium, Louisville, Kentucky, Guest Speaker: "Essential Standards Necessary in Tuberculosis Hospitals"

Dr. I. D. Bobrowitz, Otisville, New York: "Report of the Committee on Sanatorium Standards"

Dr. Allan Hurst, Denver, Colorado: "Report of the Committee on Rehabilitation"

Saturday, June 19

Council on International Affairs:

Council on Pan American Affairs, Council on European Affairs and Council on Pan Pacific Affairs

Dr. Chevalier L. Jackson, Philadelphia, Pennsylvania, Chairman, Council on Pan American Affairs, Presiding

Dr. Manual Albertal, Buenos Aires, Argentina: "Streptomycin in Tuberculosis, Clinical Experience"

Dr. Max Espinoza Galarza, Lima, Peru: "Tomography of the Larynx in Disseminated Tuberculosis"

Dr. Jorge A. Higgins, Guayaquil, Ecuador: "Experimental and Clinical Verification of the Combined Vaccines Against Smallpox and Tuberculosis"

Sir Sidney Sewell, Melbourne, Australia: "Treatment of Chest Diseases in Australia"

Dr. Basil A. Dormer, Durban, South Africa: "Treatment of Chest Diseases in South Africa"

Dr. William E. Ogden, Toronto, Canada: "The Progress of the American College of Chest Physicians in Canada"

Dr. Gustav Maurer, Davos, Switzerland: "The Progress of the American College of Chest Physicians in Europe"

Sunday, June 20

Council of Tuberculosis Committees

Dr. Jas. H. Stygall, Indianapolis, Indiana, Chairman, Presiding; Dr. I. Willner, Newark, New Jersey, Secretary

Symposium on Mass X-ray Surveys

Radiologist: Dr. Henry K. Taylor, New York, N. Y.
Pathologist: Dr. Henry C. Sweany, Chicago, Illinois
Epidemiology: Dr. R. I. Pierce, Chicago, Illinois
Chest Specialist: Dr. Allen Filek, Madison, Wisconsin

ROUND TABLE LUNCHEONS

Friday, June 18

A-1 "Tuberculosis in Children," Dr. Jay Arthur Myers, Minneapolis, Minnesota
A-2 "Treatment of Tuberculosis in Older Age Groups," Dr. Arnold Anderson, St. Petersburg, Florida
A-3 "Fungus Diseases," Dr. Alvis E. Greer, Houston, Texas
A-4 "Suppurative Diseases of the Lung," Dr. Francis M. Woods, Brookline, Massachusetts
A-5 "Relation of Race to Pulmonary Disease," Dr. Julian Lewis, Chicago, Illinois
A-6 "Chronic Cor Pulmonale Associated with Chronic Pulmonary Disease," Dr. Chauncey C. Maher, Chicago, Illinois

Saturday, June 19

B-1 "Artificial Pneumoperitoneum," Dr. Benjamin L. Brock, Downey, Illinois
B-2 "Dust Diseases," Dr. C. Howard Marcy, Pittsburgh, Pennsylvania
B-3 "Boeck's Sarcoid," Dr. David Reisner, Denver, Colorado
B-4 "Rehabilitation in Tuberculosis," Dr. Sumner Cohen, Oak Terrace, Minnesota
B-5 "Emphysema and Pulmonary Disability," Dr. George G. Ornstein, New York, N. Y.
B-6 "Atypical Pneumonias," Dr. Carl C. Aven, Atlanta, Georgia

Sunday, June 20

C-1 "Handling of Minimal Case," Dr. Herman E. Hilleboe, Albany, N. Y., and Dr. Francis Weber, Washington, D. C.
C-2 "Aerosols and Bronchial Infection," Dr. Alvin L. Barach, New York, N. Y.
C-3 "When Should Artificial Pneumothorax be Terminated," Dr. E. W. Hayes, Monrovia, California
C-4 "Rest and Early Ambulation," Dr. J. Winthrop Peabody, Washington, D. C.
C-5 "Pregnancy and Tuberculosis," Dr. Donato G. Alarcon, Mexico City, and Dr. Frederick H. Falls, Chicago, Illinois
C-6 "Dosage of Streptomycin," Dr. Emil Bogen, Los Angeles, California

The round table luncheons will be limited to 20-30 persons each. Reservations will be accepted in the order in which they are received.

Saturday, June 19

6:00 p.m. ANNUAL CONVOCATION

The College will conduct a Convocation on Saturday, June 19, when Fellowship Certificates will be awarded. The Convocation will be conducted by the Board of Regents of the College and will be open to members of the College, their families and friends. A guest speaker will address the assembly.

6:30 p.m. SOCIAL HOUR

7:30 p.m. ANNUAL PRESIDENTS' BANQUET

The Annual Presidents' Banquet will be held Saturday evening, June 19. Major General S. U. Marietta, President of the College, will deliver his presidential address, and the President-Elect, Dr. Richard H. Overholt, will be installed as President.

COLLEGE MEDAL

The second College Medal will be awarded at the Presidents' Banquet. The members of the Committee on College Awards are:

Edward W. Hayes, M.D., Monrovia, California, Chairman
Jay Arthur Myers, M.D., Minneapolis, Minnesota, Vice-Chairman
Minas Joannides, M.D., Chicago, Illinois, Secretary
Donato G. Alarcon, M.D., Mexico City, Mexico
J. Winthrop Peabody, M.D., Washington, D. C.

CLINICS

Monday, June 21

3:00 p.m. Alexian Brothers Hospital:

Demonstration of treatment of Pulmonary Tuberculosis with the use of the Lung Immobilizing Chamber (Barach).
Courtesy of Dr. Minas Joannides

Tuesday, June 22

10:00 a.m. Congress Hotel:

Demonstration of the Pneumostat, a new apparatus for constant controlled pneumothorax.
Courtesy of Dr. A. I. Breckler and Dr. Edwin R. Levine

2:00 p.m. Educational Research Hospital, University of Illinois:

Kodachrome clinic of anatomy and pathology of tracheo-bronchial tree. Courtesy of Dr. Paul H. Holinger

Wednesday, June 23

11:00 a.m. Amphitheater, Cook County Children's Hospital:

Demonstration on BCG.
Courtesy of Dr. Sol Roy Rosenthal and Dr. Erhard Loewensohn

2:00 p.m. Navy Pier and Hotel Sheraton:

Television broadcast of major surgical operation from Northwestern University Medical School.

Thursday Morning, June 24

Session on Diseases of the Chest for the Newly Created Section on Diseases of the Chest in the Scientific Assembly of the American Medical Association

Officers of the Session:

Dr. Richard H. Overholt, Brookline, Massachusetts, Chairman
Dr. J. Winthrop Peabody, Washington, D. C., Secretary

SYMPOSIUM ON STREPTOMYCIN:

Dr. Emil Bogen, Los Angeles, California:
"Laboratory Aspects of Streptomycin"
Dr. H. C. Hinshaw, Rochester, Minnesota:
"Streptomycin in Extrapulmonary Tuberculosis"
Dr. Karl H. Pfuetze, Cannon Falls, Minnesota:
"Streptomycin in the Treatment of Tuberculosis"
Dr. William Tucker, Minneapolis, Minnesota:
"Experience with Treatment of Various Forms of Tuberculosis with Streptomycin in the Veterans Administration"
Dr. Edward Beattie, Washington, D. C.:
"Use of Streptomycin in Surgical Patients"
Dr. Chester Keefer, Boston, Massachusetts:
"Antibiotics in Relation to Non-Tuberculous Chest Diseases"

Council on Pan American Affairs

Puerto Rico Chapter

The quarterly meeting of the Puerto Rico Chapter of the College was held on March 14, at the home of Dr. Philip Gorlin in Ponce.

Venezuelan Chapter

The present officers of the Venezuelan Chapter of the College are as follows:

Dr. Jose Ignacio Baldo, Caracas, President
Dr. Julio Criollo Rivas, Caracas, Vice-President
Dr. Rogelio Valladares, Caracas, Secretary-Treasurer

The new journal, "Revista Panamericana de Medicina y Cirugia del Torax," publishes news notes of College activities in Spanish, and scientific articles are published in both Spanish and English. Physicians interested in subscribing to this new journal should write to the Editor, Dr. Donato G. Alarcon, Amazonas 96, Mexico City, Mexico.

Dr. Segundo Brana Blanco, Veracruz, Mexico, has been appointed as Jefe del Departamento de Tuberculoso del Hospital Civil in Veracruz.

The officers of the Sociedad Paraense de Tisiologia, Belem, Para, Brazil, elected for the year 1948-1949 are as follows: Dr. Luiz Araujo, Presidente; Dr. Oscar Miranda, Secretario; Dr. Joao Ramos Ribeiro, Treasurer; and Dr. Geraldo Correa and Dr. Honorato Neves as members of the Review Committee.

Dr. Rene G. Mendoza, Havana, Cuba, has been elected President of the Sociedad Cubana de Tisiologia for the ensuing year.

Dr. Salvador Diaz, Santiago, Chile, has been appointed Secretary of the Santiago Chapter of the College. The Santiago Chapter is planning to hold its organizational meeting this spring.

The following physicians in the Pan American area outside of the United States, have notified the Executive Offices of the College that they are planning to attend the 14th Annual Meeting in Chicago, Illinois, June 17-20:

Dr. Antonio Acosta Velarde, Santurce, Puerto Rico
Dr. Donato G. Alarcon, Mexico City, Mexico
Dr. Manuel Albertal, Buenos Aires, Argentina
Dr. Gustavo Aldereguia, Havana, Cuba
Dr. G. C. Anglin, Toronto, Ontario, Canada
Dr. J. Garcia Arrazuria, Havana, Cuba
Dr. Jose Ignacio Baldo, Caracas, Venezuela
Dr. A. H. Baker, Calgary, Alberta, Canada
Dr. Juan J. Castillo, Havana, Cuba
Dr. Enrique Coronado Iturbide, Guatemala City, Guatemala
Dr. Ismael Cosio Villegas, Mexico City, Mexico
Dr. Arlindo de Assis, Rio de Janeiro, Brazil
Dr. Max Espinoza Galarza, Lima, Peru
Dr. Elihu J. Gutierrez, Mexico City, Mexico
Dr. I. H. Herman, Saskatoon, Sask., Canada
Dr. Jorge A. Higgins, Guayaquil, Ecuador

Dr. Miguel Jimenez, Mexico City, Mexico
Dr. G. F. Kincade, Vancouver, B. C., Canada
Dr. Harold I. Kinsey, Toronto, Ontario, Canada
Dr. Miguel C. Lascalia, Buenos Aires, Argentina
Dr. Rafael H. Leal, Guatemala City, Guatemala
Dr. Simon Marcus, Sherbrooke, P. Q., Canada
Dr. A. Marshall, Tranquille, B. C., Canada
Dr. W. K. Massey, Nelson, B. C., Canada
Dr. C. C. McLean, Kitchener, Ontario, Canada
Dr. Rene G. Mendoza, Havana, Cuba
Dr. Antonio Navarette, Havana, Cuba
Dr. William E. Ogden, Toronto, Ontario, Canada
Dr. J. Rubin, Outremont, P. Q., Canada
Dr. A. J. Pereira Rego, Rio de Janeiro, Brazil
Dr. Gumersindo Sayago, Cordoba, Argentina
Dr. Alexander C. Sinclair, St. Vital, Manitoba, Canada
Dr. Francisco Torres, Cordoba, Argentina
Dr. A. R. Valle, Seward, Alaska
Dr. Agnes M. Walker, Hamilton, Ontario, Canada

Council on European Affairs

Greek Chapter

A meeting of the Greek Chapter of the College took place in Athens on January 21, 1948. The present officers of the chapter are:

Dr. Basil Papanikolaou, Athens, President
Dr. Kyriakos Katrakis, Athens, Vice-President
Dr. Nicholas Jiannopoulos, Athens, Secretary-Treasurer

Dr. Paul Veran of Nantes, France, has been appointed Governor of the College for France.

Dr. Francis Kovats of Budapest, Hungary, has been appointed Governor of the College for Hungary.

Dr. Lopo de Carvalho of Lisbon, Portugal, has been appointed Governor of the College for Portugal.

The following physicians in Europe are planning to be present at the 14th Annual Meeting of the College in Chicago, Illinois, June 17-20:

Dr. Gustav Maurer, Davos, Switzerland
Dr. Andre Soulas, Paris, France

Council on Pan Pacific Affairs

South African Chapter Organized

The organizational meeting of the first South African Chapter took place in Cape Town on April 8, 1948. The Constitution and By-Laws were formally presented and adopted by the members present, and the following officers were elected for the chapter:

Dr. David P. Marais, Cape Town, President
Dr. T. Shrire, Cape Town, Vice-President
Dr. H. Jacob, Cape Town, Secretary-Treasurer

A photograph of the inaugural meeting was taken especially for publication in this issue of the journal, and will be found on page 464.

ORGANIZATIONAL MEETING, SOUTH AFRICAN CHAPTER, AMERICAN COLLEGE OF CHEST PHYSICIANS
CAPETOWN, SOUTH AFRICA, APRIL 8, 1948

Seated, left to right: H. O. Hofmeyr, M.D., F.C.C.P.; T. Schrire, M.D., F.C.C.P., Vice-President; D. P. Marais, M.D., F.C.C.P., President and Governor of the College for South Africa; H. H. Jacob, M.D., F.C.C.P., Secretary-Treasurer; A. Landau, M.D., F.C.C.P. — Standing, left to right: W. Phillips, M.D., F.C.C.P.; D. A. Luckoff, M.D.; H. Muller, M.D.; J. O. Marais, M.D.; W. L. Hoole, M.D., F.C.C.P.

Dr. Raman Viswanathan of New Delhi, India, has been appointed Governor of the College for India.

Dr. D. B. Rosenthal of Melbourne, Australia, is visiting in the British Isles and is planning to visit a number of other European countries during his tour.

The following physicians in the Pan Pacific area are planning to attend the 14th Annual Meeting of the College in Chicago, Ill., June 17-20:

Dr. Basil A. Dormer, Durban, South Africa
Dr. Frederick L. Giles, Honolulu, T. H.
Dr. W. Cotter Harvey, Sydney, Australia
Dr. Edmund L. Lee, Honolulu, T. H.
Dr. Alan H. Penington, Melbourne, Australia
Dr. Forrest J. Pinkerton, Honolulu, T. H.
Dr. Karun Sankar Ray, Calcutta, India
Sir Sidney Sewell, Melbourne, Australia
Dr. S. H. Tsai, China

College Chapter News

ARIZONA CHAPTER

The Arizona Chapter of the College will hold its annual meeting on May 19 at the Westward Ho Hotel, in Phoenix. The following program will be presented:

"Bronchography," Howell Randolph, M.D., F.C.C.P., Phoenix Arizona
"Exploratory Thoracotomy in Obscure Thoracic Disease," John B. Grow, M.D., F.C.C.P., Denver, Colorado
"The Use of the Laboratory in Controlling the Treatment of Tuberculosis," C. Richard Smith, M.D., Los Angeles, California

The scientific program will be followed by a luncheon and business meeting of the chapter. The guest speaker at the luncheon will be Dr. J. Dewey Bisgard of Omaha, Nebraska, who will discuss "Resection for Non-Tuberculous Pulmonary Disease."

CALIFORNIA CHAPTER

At the annual meeting of the California Chapter of the College, held on April 10, the following officers were elected to office for the ensuing year:

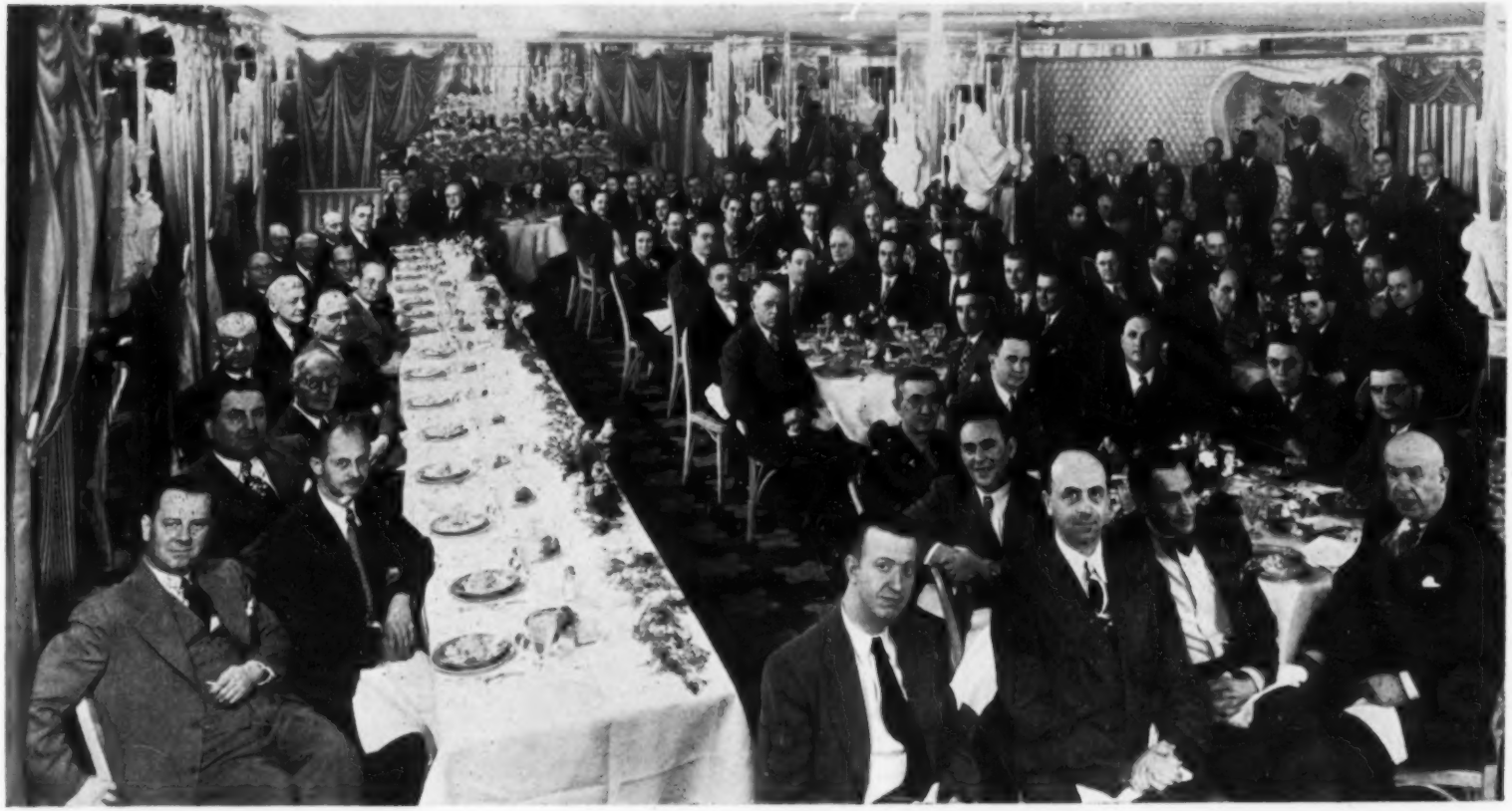
Cabot Brown, M.D., San Francisco, President
Lyman A. Brewer, III, M.D., Los Angeles, Vice-President
Seymour M. Farber, M.D., San Francisco, Secretary-Treasurer

INDIANA CHAPTER

The Indiana Chapter of the College held a luncheon meeting at the Columbia Club, Indianapolis on Sunday, April 18. A scientific program followed, presided over by Dr. J. V. Pace, F.C.C.P., New Albany, President of the chapter. The following papers were presented:

"Value of Planigrams in Chest Conditions," Phillip Cohn, M.D., New Albany

DINNER, FIRST ANNUAL POSTGRADUATE COURSE IN DISEASES OF THE CHEST



Sponsored by the Council on Postgraduate Medical Education of the American College of Chest Physicians and the Laennec Society of Philadelphia. Barclay Hotel, Philadelphia, Pennsylvania, Monday, March 15,1948

"Pleural Effusions,"
Edward W. Custer, M.D., F.C.C.P., South Bend
"Intrathoracic Tumors and Cysts,"
John V. Thompson, M.D., F.C.C.P., Indianapolis

The meeting closed with an X-ray Conference at which many interesting films were shown.

MICHIGAN CHAPTER

The Michigan Chapter of the College held its spring meeting in Detroit on March 30, at the Detroit Tuberculosis Sanatorium. Dinner was served and following this, Dr. Richard H. Overholt, Brookline, Massachusetts, President-Elect of the American College of Chest Physicians, the guest speaker, was introduced. Dr. Overholt spoke on "Significance of Segmental Resection in Thoracic Diseases," which was followed by an interesting discussion.

A business meeting and election of officers was held. The Fall meeting was discussed, which will be held in September in conjunction with the Michigan State Medical Society meeting in the Book Cadillac Hotel, Detroit. The 1947 officers of the chapter were re-elected as follows:

Arthur R. Young, M.D., Pontiac, President
William P. Chester, M.D., Detroit, Vice-President
Cletus J. Golinvaux, M.D., Monroe, Secretary-Treasurer

The Michigan Chapter expressed its thanks to the Staff of the Detroit Tuberculosis Sanatorium for their hospitality.

MISSOURI CHAPTER

The new officers of the Missouri Chapter of the College recently elected for the year 1948 are as follows:

George D. Kettelkamp, M.D., Koch, President
Alfred Goldman, M.D., St. Louis, Vice-President
A. J. Steiner, M.D., St. Louis, Secretary-Treasurer (re-elected)

The following committees have been appointed by the President:

Membership Committee:
Charles A. Brasher, M.D., Mt. Vernon, Chairman
Jesse E. Douglass, M.D., Webb City
Louis Levin, M.D., Excelsior Springs

Medical Education Committee:
James L. Mudd, M.D., St. Louis, Chairman
Alfred Goldman, M.D., St. Louis
Sam H. Snider, M.D., Kansas City

Program Committee:
Herbert L. Mantz, M.D., Kansas City, Chairman
William W. Buckingham, M.D., Kansas City
Bernard Friedman, M.D., Koch

NEW JERSEY CHAPTER

The New Jersey Chapter of the College met at Haddon Hall, Atlantic City, on April 28, in connection with the annual meeting of the Medical Society of New Jersey. The scientific program was presented in the Section on Diseases of the Chest in the state medical society on that day. The Medical Society of New Jersey is the first state medical society

FIRST ANNUAL POSTGRADUATE COURSE IN DISEASES OF THE CHEST SPONSORED BY THE COUNCIL ON POSTGRADUATE MEDICAL EDUCATION, AMERICAN COLLEGE OF CHEST PHYSICIANS AND THE LAENNEC SOCIETY OF PHILADELPHIA

WARWICK HOTEL, PHILADELPHIA, PENNSYLVANIA, MARCH 15-20, 1948

Some of the physicians and instructors who participated in the First Annual Postgraduate Course in Diseases of the Chest

to establish a Section on Diseases of the Chest in their scientific assembly.

Following the scientific section, the New Jersey Chapter held its annual meeting and dinner at the hotel. The following officers were elected for the ensuing year:

Paul K. Bornstein, M.D., Asbury Park, President
Homer H. Cherry, M.D., Paterson, First Vice-President
Bertram S. Pollak, M.D., Jersey City, Second Vice-President
Benjamin P. Potter, M.D., Jersey City, Secretary-Treasurer

NORTH MIDWEST CHAPTER

The North Midwest Chapter will hold its annual meeting on June 7 at the Radisson Hotel, Minneapolis, at the time of the annual meeting of the Minnesota State Medical Association. There will be a luncheon for College members and guests at the Radisson Hotel at 12:00 noon, at which time a business meeting of the chapter will be held. A scientific program will follow at 2:00 p. m. The program to be presented is given below:

Jay Arthur Myers, M.D., F.C.C.P., Minneapolis, Moderator

"Dangers of Delay in the Diagnosis of Indeterminate Pulmonary Lesions,"
Allen Good, M.D., Rochester
Discussors: E. P. J. Fenger, M.D., Oak Terrace,
Thomas Lowry, M.D., Minneapolis

"Some Experiences with Streptomycin in the Treatment of Tuberculosis in the Veterans Administration,"
William B. Tucker, M.D., Minneapolis
Discussors: Russell Frost, M.D., F.C.C.P., St. Paul,
Karl H. Pfuetze, M.D., F.C.C.P., Cannon Falls

"Nebulization Therapy for Pulmonary Diseases,"
Arthur Olsen, M.D., Rochester
Discussors: Jay Arthur Myers, M.D., F.C.C.P., Minneapolis,
G. A. Hedberg, M.D., F.C.C.P., Nopeming

"Diagnosis of Peripheral Lung Tumors Observed in Chest Survey,"
David Sharp, M.D., F.C.C.P., and
Robert Nord, M.D., Minneapolis
Discussors: William Roemmich, M.D., Minneapolis,
Sumner S. Cohen, M.D., F.C.C.P., Oak Terrace

OHIO CHAPTER

At the annual meeting of the Ohio Chapter, held in Cincinnati on March 31, the following officers were elected for the year 1948-1949:

William J. Habeeb, M.D., Springfield, President
Garry G. Bassett, M.D., Lakewood, Vice-President
Everett F. Conlogue, M.D., Dayton, Secretary-Treasurer

Dr. Louis Mark, Columbus, made a motion at the business meeting of the Ohio Chapter, that the chapter donate $100.00 to the Research Fund that has been set up by the national organization, this money to be taken from the Ohio Chapter Treasury. The motion was seconded by Dr. William Habeeb. It was carried unanimously and the Treasurer was directed to send the contribution to the Executive offices of the College.

POTOMAC CHAPTER

The following officers of the Potomac Chapter were elected at the chapter meeting in Baltimore, Maryland, on April 26:

Otto C. Brantigan, M.D., Baltimore, Maryland, President
Edgar W. Davis, M.D., Washington, D. C., Vice-President
Milton Kress, M.D., Towson, Maryland, Secretary-Treasurer

TEXAS CHAPTER

The Texas Chapter held its annual meeting in Houston on April 26, and the following officers were elected for the year 1948-1949:

Robert B. Homan Jr., M.D., El Paso, President
Elliott Mendenhall, M.D., Dallas, First Vice-President
Jesse B. White, M.D., Amarillo, Second Vice-President
Charles J. Koerth, M.D., Kerrville, Secretary-Treasurer

NEW YORK STATE CHAPTER

On the evening of May 20 an informal dinner of the New York State Chapter of the College will be held at the Hotel Pennsylvania, New York City. A business meeting and election of officers will be held which is to be followed by an address by Major General S. U. Marietta, President of the American College of Chest Physicians.

In the afternoon of May 20 a symposium of Primary Carcinoma of the Lung has been arranged by Dr. Joseph J. Witt, Chairman, Session on Chest Diseases of the Medical Society of the State of New York. It is as follows:

"Diagnosis,"
John Hayes, M.D., Saranac Lake, New York
"Pathological Aspects,"
Oscar Auerbach, M.D., New York, N. Y.
"Role of the Bronchoscopist in Diagnosis and Treatment,"
Louis Clerf, M.D., F.C.C.P., Philadelphia, Pennsylvania
"Treatment,"
Richard H. Overholt, M.D., F.C.C.P., Brookline, Massachusetts

ILLINOIS CHAPTER

The annual meeting of the Illinois Chapter was held at the Palmer House, Chicago, on Monday evening, May 20. A business meeting and election of officers was held and Dr. Roy A. Wolford, Acting Assistant Medical Director for Professional Services, Veterans Administration, Washington, D. C., was the guest speaker. His subject was "Program of the Veterans Administration on Diseases of the Chest."

The new officers elected for the chapter are as follows:

Kenneth G. Bulley, M.D., Aurora, President
Darrell H. Trumpe, M.D., Springfield, Vice-President
Edwin R. Levine, M.D., Chicago, Secretary-Treasurer

The following members of the College presented papers at the Annual Meeting of the Illinois State Medical Society, Chicago, May 10, 11 and 12:

"The Present Status of Streptomycin,"
Dr. Karl H. Pfuetze, Cannon Falls, Minnesota
"The Physician's Responsibility Toward the Hard of Hearing and Deafened,"
Dr. Francis L. Lederer, Chicago, Illinois

"The Treatment of Pneumonia with Single Daily Intramuscular Injections of Penicillin and Wax,"
Dr. Italo Volini, Chicago, Illinois

"Early Diagnosis of Cancer of the Stomach,"
Dr. Leo G. Rigler, Minneapolis, Minnesota

"Recent Techniques in the Management of Bronchial Infections,"
Dr. Edwin R. Levine, Chicago, Illinois

"Detection of Various Chest Lesions by Mass X-ray Surveys,"
Dr. Dan Morse, Peoria, Illinois

Dr. Paul H. Holinger, Chicago, Illinois, presided at the session on Monday afternoon, May 10, and at the Section on Eye, Ear, Nose and Throat on Tuesday morning, May 11. Dr. S. A. Levinson, Chicago, Illinois, presided at the Section on Pathology on Tuesday afternoon, May 11.

College News Notes

Harry C. Warren, M.D., F.C.C.P., San Francisco, California, Second Vice-President of the College, has been elected President of the California Tuberculosis and Health Association, and Edward W. Hayes, M.D., F.C.C.P., Monrovia, California, a Past-President of the College, has been elected as Vice-Chairman of the association.

Paul H. Holinger, M.D., F.C.C.P., Chicago, Illinois, was elected President of the American Broncho-Esophagological Association at their 29th Annual Meeting held in Atlantic City in April. Dr. Holinger formerly served as Secretary-Treasurer of the Association.

Alvan L. Barach, M.D., F.C.C.P., George F. Herben, M.D., F.C.C.P., James H. Cullen, M.D., F.C.C.P., and Chesmore Eastlake, M.D., New York, New York, will present a paper entitled "Clinical Recovery in Advanced Pulmonary Tuberculosis Induced by Total Lung Rest in the Immobilizing Lung Chamber," in the Scientific Assembly of the American Medical Association in June. Dr. Barach will also have a booth in the scientific exhibit of the American Medical Association on "Immobilizing Lung Chamber Therapy in Pulmonary Tuberculosis."

Seymour M. Farber, M.D., F.C.C.P., San Francisco, California, presented a paper on "Bronchogenic Carcinoma" before the annual meeting of the California Medical Association and the American College of Physicians which took place in San Francisco in April.

R. S. Anderson, M.D., F.C.C.P., Erie, Pennsylvania, has been appointed to succeed Benjamin L. Brock, M.D., F.C.C.P., Downey, Illinois, as Chairman of the Council of Medical Directors and Superintendents of Tuberculosis Hospitals and Sanatoria of the College. Dr. Anderson formerly served as Secretary of the Council. E. W. Custer, M.D., F.C.C.P., South Bend, Indiana, has been appointed to the office of Secretary of the Council. Dr. Brock recently joined the staff of the Veterans Hospital at Downey, Illinois.

J. Edmond Bryant, M.D., F.C.C.P., Evanston, Illinois, recently returned from a visit to Haiti, where he had an opportunity to meet with other members of the College.

Obituaries

PROFESSOR ENRIQUE FINOCHIETTO

1881 - 1948

In the history of South American surgery, Professor Enrique Finochietto represents the figure of greatest scientifical prestige. His creative talent and investigator's soul brought him to the perfecting of many surgical techniques, animated with an enthusiasm only comparable to the greatest masters of all times.

Modest and unassuming he never obtruded his personality or exploited his merits or achievements as claim for priority, but rather referred to himself in an impersonal way, and allowed the facts to speak for themselves. It is not only because he was one of the greatest surgeons of his generation that he deserves special homage, nor even because of the great debt which surgery owes him, but more especially for his fundamental contributions to the enlargement of the field of surgery and to the elevation of its standards to a nobler height.

He created methods in surgery, which he taught using continuously his own unexcelled skill as an example in the operating room. He was always ready to suggest new ideas, urging the younger men who surrounded him, to explore new paths, remaining unto the end the inspirer and the undisputed chief of a group of brilliant men.

Professor Enrique Finochietto was born the 13th of March 1881. He graduated from the Medical School of the University of Buenos Aires with highest honours. He was appointed surgeon on the staff of the Rawson Hospital. He went abroad several times to Europe and the United States, to study with the eminent men in the field of surgery. It was on one of these trips that the French Government conferred upon him the honorary title of the Officer of the Legion of Honor, and later he received the medal of the Red Cross of Paris and the Golden Medal given to him for his surgical services to the soldiers.

He won the appointment of surgeon in chief at the Rawson Hospital through competitive examination and later became professor of surgery in the School of Medicine of the University of Buenos Aires.

He was president of several surgical congresses and a member of institutions of world prestige, enjoying the respect and admiration of his European and American colleagues. At the conclusion of thirty years of scientifical work his pupils offered him a golden book with a series of original papers about subjects studied under his tutelage, giving him in such a way sincere homage.

The Finochietto brothers, who represent in South America, what the Mayo brothers represent in the United States, are publishing a fundamental treatise of surgery received with keen attention in the medical centers all over the world. This "magnus opere" will be terminated by Professor Ricardo Finochietto, twin personality of his eminent brother with whom he was always the most admirable co-worker.

With his illustrious brother Prof. Ricardo Finochietto, who survives him, they have created a surgical school of the greatest renown which includes the most brilliant group of surgeons of the Argentine Republic.

The Finochietto school of surgery through the Surgical Sessions for Postgraduates, the most qualified teaching institution of the continent created under the inspiration of Enrique Finochietto and directed by Ricardo Finochietto, has become the Mecca of the South American Surgery where surgeons come in ever increasing numbers. The name Finochietto represents a symbol of surgery at its best in South America, being at the same time synonymous with the art of surgery in the highest conception of the term.

Dr. Enrique Finochietto attacked every problem that came before him at the foundation. He was not satisfied with the superstructure and would not rest until assured of the security of its base.

His artistic sense, his wide culture, his manual dexterity, his absolute assurance, his tenacity of purpose all made professor Enrique Finochietto, whose death occurred the 17th of February 1948, in the middle of his best creative capacity, one of the greatest surgeons of the century.

Considerado universalmente como una figura del más elevado prestigio científico el Prof. Enrique Finochietto representa en la historia de la cirujía sud-americana el valor estelar de mayor magnitud.

Su alma de investigador lo llevó al terreno de la especulación científica y su talento creador lo condujo a la perfección de la técnica quirúrgica, animándolo para ello un entusiasmo parangonable solo al de los grandes maestros de todas las épocas.

Dotado de un espíritu crítico profundo su labor ha sido trascendental y la simiente que ha ido esparciendo ha dado fruto fecundos, reflejándose en los innumerables discípulos a los cuales su obra seguirá iluminando en el futuro. Creador de metodos y de sistemas quirúrgicos los aplicaba enseñando, constantemente dando la lección incomparable de su propia labor en la sala de cirujía, sin dar a su ímproba tarea la mínima suntuosidad espectacular.

No menos importante que sus fundamentales contribuciones a la cirujía fueron el resultado del impacto de su personalidad, sobre los numerosós discípulos que ha formado, inspirándoles constantemente un anhelo de superación integral.

Había nacido el Dr. Enrique Finochietto el 13 de Marzo de 1881. Cursó sus estudios universitarios en la Facultad de Medicina de Buenos Aires donde obtuvo su título de doctor en medicina con la actuación mas sobresaliente.

Nombrado cirujano del Hospital Rawson efectuó repetidos viajes de estudio a Europa y Estados Unidos para apreciar los procedimientos de los grandes maestros de la época. En uno de estos viajes, el gobierno de Francia le confirió el título de Oficial de la Legión de Honor y posteriormente recibió la medalla de la Cruz Roja de París y la de oro de la Guerra otorgada por su atención a los soldados.

Obtuvo por concurso los cargos de jefe de Cirujía del Hospital Rawson y luego Professor de Cirugía en la Escuela de Medicina de la Universidad de Buenos Aires.

Presidió congresos y reunione de especialistas, fue miembro de las institucion de mayor prestigio mundial, gozando del respeto y admiración de sus colegas europeos y americanos de más alta jerarjuía.

Cuando cumplió 30 años de labor científica sus discípulos reunieron en un libro de Oro una serie de trabajos originales sobre temas estudiados bajo la dirección del Maestro, rindiéndole en esa forma un homenaje justiciero.

Con su ilustre hermano, el Profesor Ricardo Finochietto que lo sobrevive, han creado la escuela quirúrgica del mayor prestigio, que agrupa al nucleo de cirujanos más brillantes del continente. Los hermanos Finochietto, que representan en Sud-America, lo que los hermanos Mayo significaron en los Estados Unidos, han iniciado la publicación de una obra fundamental y consagratoria, recibida con interés muy vivo en los medios científicos universales.

Esta obra ciclópea será terminado por el Profesor Ricardo Finochietto, personalidad gemela de su eminente hermano, del cual fuera siempre el colaborado admirable.

La escuela Finochietto gracias a las sesiones Quirúrgicas para Graduados, institución docente mas calificada del continente, creado bajo la inspiración de Enrique Finochietto, ordenadas y dirigidas por Ricardo Finochietto, se han transformado en la Meca de la Cirujía Sud Americana, donde seguirán concurriendo en forma creciente, los cirujanos de la Republica Argentina y de todos los países del continente a perfeccionar sus conocimientos quirúrgicos.

El nombre Finochietto representa un simbolo en la cirugia del continente siendo a su vez sinónimo de arte quirúrgico en la mas elevada acepción del termino.

Su elevado sentido artístico, su delicadeza de espiritu, su cultura integral, su agilidad mental, su extraordinaria habilidad técnica, puestas al servicio de una disciplina tenaz, todas estas cualidades hicieron del Profesor Enrique Finochietto, desaparecido el 17 de Febrero de 1948 en la plenitud de su capacidad creadora, uno de los grandes cirujanos del siglo.

David Fairman, M.D.

LEON GILBERT WOODFORD

1882 - 1947

Dr. Leon Gilbert Woodford was born in Coal Creek, Colorado, on June 19, 1882. He graduated from the University of Colorado in 1909 and in 1910 went to Everett, Washington where he practiced as physician and surgeon for 37 years, specializing in diseases of the chest. Dr. Woodford was one of the men primarily responsible for the founding of Aldercrest Sanatorium and for many years was medical director of that institution. He also served on the staff of the Sisters of Providence Hospital and the Everett General Hospital.

Dr. Woodford was a Life Member and Fellow of the American College of Chest Physicians, a member of the Snohomish County Medical Society and the Washington State Medical Association. Through the years Dr. Woodford contributed many articles to the journal "Northwest Medicine" and presented papers at a number of medical meetings.

Dr. Woodford died December 28, 1947 after a short illness. He is survived by his widow, Mrs. Catherine Woodford of Everett, Washington, a daughter and a grandson.

John E. Nelson, M.D., Governor for Washington.